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Robert W. Hobson II *Editors*

Vascular Surgery

Cases, Questions and Commentaries

Second Edition


Springer

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This book is dedicated to the memory of Polychronia Geroulakos, Senior Sister in the Geroulakos Clinic in Sparta, Greece. For almost 40 years she looked tirelessly after the patients of this institution in an exemplary manner leaving a legacy of high standards.



Union Européenne des Médecins Spécialistes

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Barcelona, March 2006

Vascular Surgery is a discipline that deals with one of the true plagues of the 20th century. Moreover, atherothrombosis will continue to be the main cause of death in the near future.

New developments in the investigation, and endoluminal treatment of vascular disease have recently attracted significant publicity from the mass media and patient groups, and have significantly changed the management of the vascular patient.

The provision of a high quality vascular service is closely linked with the need to give residents an appropriate training and to further introduce Vascular Surgery as an outstanding specialty.

The book, “Vascular Surgery; Cases, Questions and Commentaries”, by Mr. Geroulakos, Prof Hero van Urk and Dr. R W Hobson II, will indeed contribute to a better understanding of Vascular Surgery as a specialty that deals with the pathology of arteries, veins and lymphatics. The experience and the teaching capabilities of the authors are unquestionable.

This book, being so comprehensive, enhances the idea of considering Vascular Surgery as an independent entity from other specialties. Before achieving adequate competence to deal with the variety of cases shown in the book, the need for an appropriate training is obvious. Besides, the present text will help candidates to better prepare for the EBSQ-Vasc examination. The book utilises a time proven concept for teaching by questions and answers based on real problems, an essential part of CME. The book proposes learning following the Socratic method, by exercising our mind rather than reading told facts. On the other hand, it may improve our clinical practice and care of our vascular patients, as it incites Continuous Professional Development as a step forward in CME.

The European Board of Vascular Surgery congratulates the authors for their initiative and gladly endorses the book.

Marc Cairols
Secretary General
UEMS Section and Board of Vascular Surgery

Foreword to the First Edition

This book is rather unique among textbooks in vascular surgery. Most cover the surgical management of vascular diseases, in whole or in part, in standard textbook fashion, with the text organized to cover the topics methodically in a didactic manner, and supported by tables, illustrations and references. Others have special purposes, such as atlases on technique or algorithm based books on decision-making. All have their place, but if the educational goals are training of the young surgeon, self-assessment and continuing medical education for the practitioner or preparation for oral examination, this book fills a special need, and fills it very well by breaking away from the didactic approach.

It has long been recognized by educators that retention of knowledge, i.e. true learning, are much better achieved using the Socratic method of questions and answers, as opposed to simply reading or being told facts. In this book this approach is developed and presented in a very effective manner. In each "chapter", one is presented with a case report representing a real life scenario. The case reports-scenarios in this book together cover most of vascular surgery experience. Following the case report, one is presented with questions and answers based on various aspects of the case, forcing the reader to commit to an answer. Whether the answer is right or wrong is not critical, in fact getting a wrong answer may be more beneficial in terms of correcting knowledge and retaining information. The commentary and conclusions that follow analyze the choice of answers, correct and incorrect, and discuss them in concise, authoritative detail, many of which are truly "pearls of information". The conclusion then summarizes the current state of knowledge on the clinical issues under consideration. Numerous references are included. Together, these components constitute one of the most effective vehicles for self-education in vascular surgery today. Importantly, all aspects of management are covered: diagnostic evaluation and appropriate treatment, whether it is non-operative or interventional, endovascular or open surgery.

To accomplish their goals the editors have gathered together a large number of experienced contributors, many well-known for their special areas of interest within vascular surgery, reflected in the contributions they make to this book. As such, the book should be useful to future and practicing vascular surgeons all over the world. It is full of statements covering most of the current state of knowledge in vascular surgery, and it does so in an entertaining and effective manner.

Robert B. Rutherford MD, FACS, FRCS
Emeritus Professor of Surgery
University of Colorado

Preface to the First Edition

This book is a unique collection of real life case histories written by experts that highlight the diversity of problems that may be encountered in vascular surgery. Each case scenario is interrupted by several questions that aim to engage the reader in the management of the patient and to give him the opportunity to test his knowledge. The comments reflect to as much as possible the principles of evidence based medicine and provide the answers to the questions.

Several chapters are authored by individuals that contributed to the development of innovations in the management and prevention of vascular disease and are of interest for both the vascular trainee and the experienced vascular specialist.

The goal of this book is to help vascular trainees review for Board and other examinations as well as to provide vascular surgeons who wish to expand or refresh their knowledge with an update and interactive source of information relevant to case scenarios that could be encountered in their practice.

The European Boards in Vascular Surgery is a relatively new examination. Although the American Boards in Vascular Surgery were established many years earlier, there are no "dedicated" guides to cover the needs of these examinations. We hope that our book will provide a helpful hand that does not come from the standard text books, but directly from daily practice and therefore contains a high content of "how to do it" and "why we do it". The references show the close relation between daily practice and "evidence based" practice, and we hope the two are not too different.

We would like to thank all the authors who have contributed generously their knowledge and time to this project.

George Geroulakos
Hero van Urk
Keith D Calligaro
Robert Hobson II

Preface to the Second Edition

The authors' principal objective in the first edition of *Vascular Surgery* was the presentation of the principles of vascular and endovascular surgery through interactive real life clinical scenarios. The success of the first edition has been gratifying. We have received many suggestions for additions and changes from vascular trainees, specialists, and teachers at various institutions in Europe, the United States, and other parts of the world. These comments have been well received and have been important in improving and expanding the second edition. We wish to acknowledge our appreciation and gratitude to our authors and publishers.

George Geroulakos
Hero van Urk
Robert W Hobson II
London, Rotterdam, and New Jersey
March 2006

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1. Preoperative Cardiac Risk Assessment and Management of Elderly Men with an Abdominal Aortic Aneurysm

Don Poldermans and Jeroen J. Bax

A 72-year-old male presented with an abdominal aortic aneurysm. He had a history of chest pain complaints and underwent percutaneous transluminal coronary angioplasty (PTCA) 6 years ago. After the PTCA procedure he had no chest pain symptoms until 2 years ago. The chest pain complaints are stable and he was able to perform moderate exercise, such as a round of golf, in 4.5 hours. Physical examination showed a friendly man, with blood pressure 160/70 mm Hg and pulse 92 bpm. Examination of the chest revealed no abnormalities of the heart. Palpation of the abdomen showed an aortic aneurysm with an estimated diameter of 7 cm. The patient was referred to the vascular surgeon. Blood test showed an elevated fasting glucose of 10.0 mmol/l and low-density lipoprotein (LDL) cholesterol of 4.1 mmol/l. Electrocardiography showed a sinus rhythm and pathological Q-waves in leads V1–V3, suggestive of an old anterior infarction.

Question 1

Which of the following statements regarding postoperative outcome in patients undergoing major vascular surgery is correct?

A. Cardiac complications are the major cause of perioperative morbidity and mortality.

B. Perioperative myocardial infarctions are related to fixed coronary artery stenosis in all patients.

C. Perioperative cardiac events are related to a sudden, unpredictable progression of a nonsignificant coronary artery stenosis in all patients.

D. Perioperative cardiac complications are related to both fixed and unstable coronary artery lesions.

This patient experienced angina pectoris in the past. He was successfully treated with a PTCA procedure, but recently angina pectoris reoccurred. Because of the

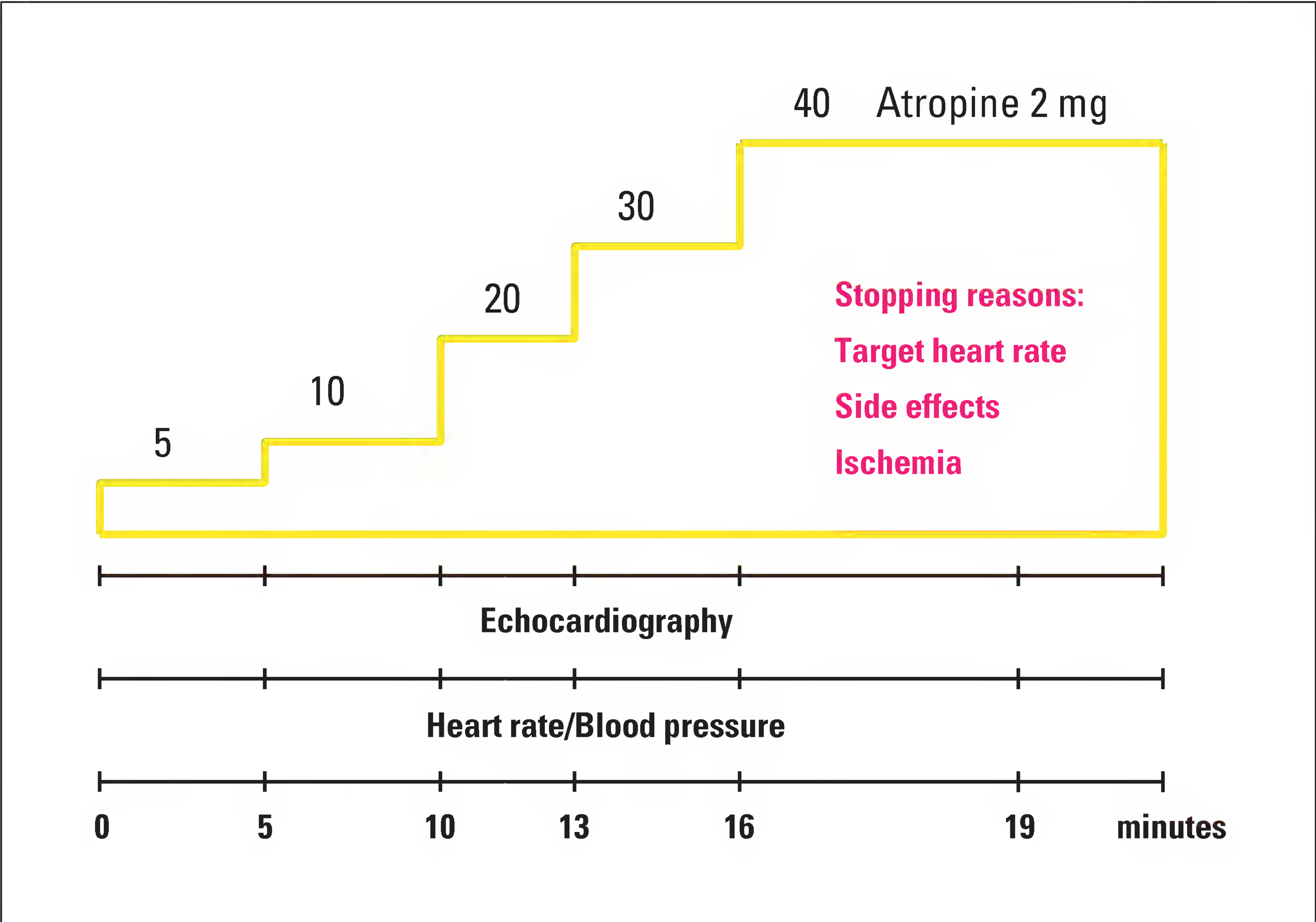


Fig. 1.1. The normal stress protocol, with increasing doses of dobutamine and test endpoints.

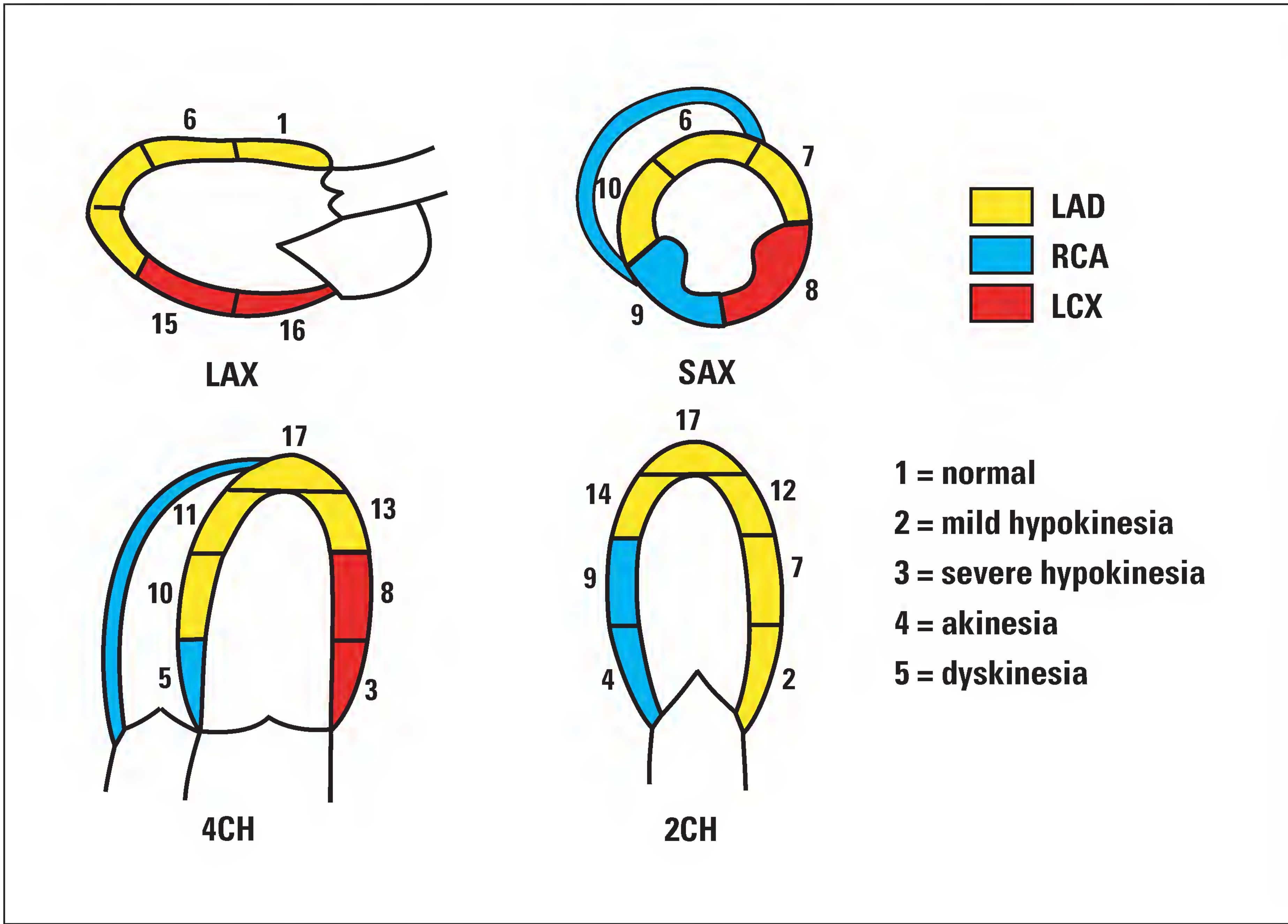


Fig. 1.2. The scoring of the left ventricle for wall motion abnormalities. LAX, long axis; SAX, short axis; 4CH, four chambers; 2CH, two chambers; LAD, left anterior descending artery; RCA, right coronary artery; LCX, left circumflex artery.

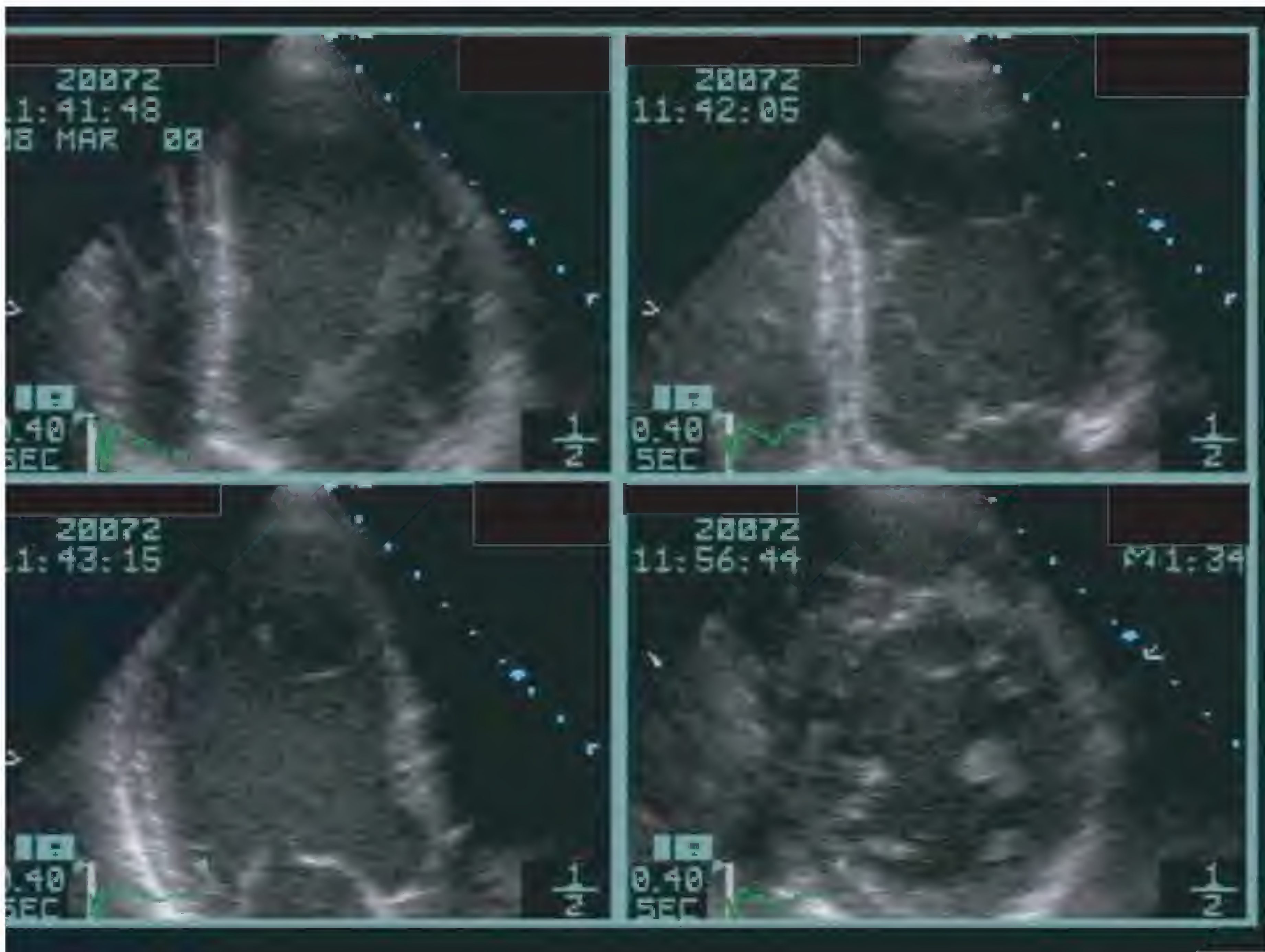


Fig. 1.3. An example of a normal resting echocardiogram, showing respectively, apical views and one short-axis view.

multiple risk factors and the planned high-risk surgery a dobutamine stress echocardiography was performed. Fig. 1.1 shows the normal stress protocol, with increasing doses of dobutamine and test endpoints. In Fig. 1.2 the scoring of the left ventricle for wall motion abnormalities is shown. Fig. 1.3 is an example of a normal resting echocardiogram, showing respectively, apical views and one short-axis view. In Fig. 1.4, the different stages of the stress test are shown for the apical four-chamber view: rest, low-dose dobutamine, peak dose dobutamine, and recovery. As indicated by arrows, the posterior septum shows an outward movement during peak stress, suggesting dyskinesia, and myocardial ischemia of the posterior septum.

Question 2

Postoperative outcome in patients undergoing major vascular surgery has been improved in those taking beta-blockers and statins. Medical therapy may reduce the need for additional preoperative testing for coronary artery disease as the incidence of perioperative cardiac mortality is reduced to less than 1 percent, and may even reduce the indications for preoperative coronary revascularization.

A. Beta-blockers are associated with a reduced perioperative cardiac event rate in patients undergoing vascular surgery, both in retrospective and prospective studies.

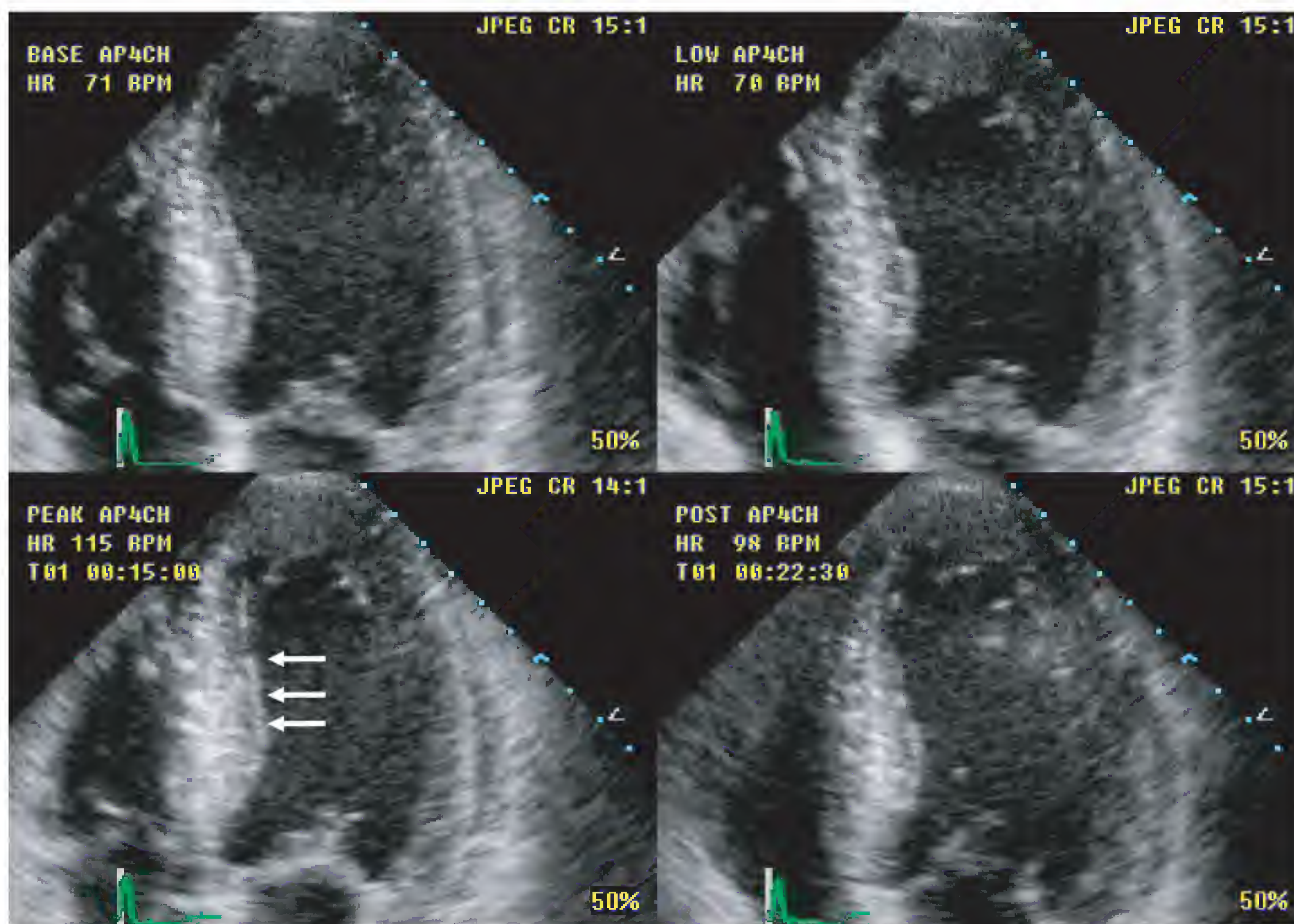


Fig. 1.4. The different stages of the stress test of the apical four-chamber view, rest, low-dose dobutamine, peak dose dobutamine, and recovery. As shown and indicated with arrows, the posterior septum shows an outward movement during peak stress, suggestive of dyskinesia, and also myocardial ischemia of the posterior septum.

B. Statin use is associated with an improved postoperative outcome.

C. Statin use is not associated with an increased incidence of perioperative myopathy.

D. Beta-blockers and statins are independently associated with an improved postoperative outcome.

Question 3

Preoperative beta-blocker therapy is widely used. However, the dose and duration of preoperative therapy is uncertain.

A. Beta-blockers should be started preferably 30 days prior to surgery.

B. Beta-blockers should be initiated several hours before surgery.

C. Heart rate control should be aimed at a heart rate between 90 and 100 bpm.

D. Heart rate control should be aimed at a heart rate between 60 and 70 bpm.

In this patient beta-blockers were started 6 weeks before surgery. Starting dose of bisoprolol was 2.5 mg; the dose was increased to 5.0 mg to obtain a resting heart between 60 and 70 bpm.

Question 4

Perioperative statin therapy has recently been introduced to improve postoperative outcome.

A. Statins improve postoperative outcome by reducing the cholesterol level.

B. Withdrawal of perioperative statin therapy is associated with an increased perioperative cardiac event rate.

C. Perioperative statin use is associated with an increased incidence of myopathy.

D. Perioperative statin use is associated with a reduced perioperative cardiac event rate in vascular surgery patients only.

Statins were prescribed in this patient, Lescol XL 80 mg daily, at the same time as beta-blockers were introduced.

Question 5

Preoperative coronary revascularization seems to be an attractive option to improve not only direct postoperative outcome in high-risk patients but also long-term survival after surgery.

A. Preoperative coronary revascularization improves postoperative outcome in all patients with significant coronary artery disease prior to major vascular surgery.

B. Preoperative coronary revascularization in patients with one- or two-vessel disease is not associated with an improved postoperative outcome compared to patients receiving medical therapy.

C. Preoperative coronary revascularization is associated with an improved 2-year outcome compared to medical therapy.

D. Patients with proven coronary artery disease who are treated medically are at increased risk of late coronary revascularization after surgery. After late revascularization, long-term outcome is similar to that with revascularization prior to surgery.

This 72-year-old male had multiple cardiac risk factors: elderly age, angina pectoris, diabetes mellitus, and a previous MI. He underwent a noninvasive stress test, dobutamine stress echocardiography, which showed myocardial ischemia, suggesting left anterior descending artery (LAD) disease. Beta-blockers and statins were prescribed and continued during surgery. Surgery was uneventful; after 2 years angina pectoris complaints increased and a PTCA procedure was successfully performed on the LAD.

Commentary

Cardiac complications are the major cause of perioperative morbidity and mortality, which may occur in 1–5 percent of unselected patients undergoing major

vascular surgery [1]. **[Q1: A]** This high frequency of cardiac complications is related to the high prevalence of coronary artery disease; 54 percent of patients undergoing major vascular surgery have advanced or severe coronary artery disease and only 8 percent of patients have normal coronary arteries [2]. Perioperative cardiac complications are equally caused by prolonged myocardial ischemia or by coronary artery plaque rupture with subsequent thrombus formation and coronary artery occlusion [1, 3]. **[Q1: B, C, D]** Prolonged perioperative myocardial ischemia usually occurs from either increased myocardial oxygen demand or reduced supply, or from a combination of the two. There are several perioperative factors that can increase myocardial oxygen demand including tachycardia and hypertension resulting from surgical stress, postoperative pain, interruption of beta-blocker use, or the use sympathomimetic drugs. Decreased oxygen supply, on the other hand, can occur as a result of hypotension, vasospasm, and anemia, hypoxia or coronary artery plaque rupture. Beta-blockers primarily reduce myocardial oxygen demand, while statins may prevent coronary artery plaque rupture. **[Q2: A, B]**

Beta-Adrenergic Antagonists

Several retrospective and prospective clinical trials have shown that perioperative use of beta-blockers is associated with reduction in the incidence of postoperative myocardial ischemia, nonfatal myocardial infarction and cardiac death [4–6]. **[Q2: A]** The majority of these studies were small in sample size, and the studies were designed to explore the protective effect of beta-blockers for the reduction of perioperative myocardial ischemia. To overcome the limitations of these studies two randomized clinical trials addressed the issue of perioperative use of beta-blockers for the prevention of cardiac death and myocardial infarction. Mangano et al. [7] studied the effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery including vascular surgery. The investigators enrolled and randomized 200 patients to atenolol (given intravenously before and immediately after surgery and orally thereafter for the duration of hospitalization) or placebo. No difference was observed in 30-day mortality but mortality was significantly lower at 6 months following discharge (0% vs. 8 %, $p < 0.001$), over the first year (3% vs. 14%, $p = 0.005$), and over 2 years (10% vs. 21%, $p = 0.019$). The apparent lack of a perioperative cardioprotective effect of atenolol in this study was probably related to the small sample size, and the fact that patients at low risk for cardiac complications were studied. In a more recent study, Poldermans et al. [8] clearly demonstrated the cardioprotective effect of perioperative beta-blocker use for the reduction of perioperative cardiac death and myocardial infarction in high-risk patients undergoing major vascular surgery. In total, 112 high-risk vascular patients were selected using a combination of cardiac risk factors and positive results on dobutamine stress echocardiography. Patients were then randomly assigned to standard care or standard care with bisoprolol use. Bisoprolol was started at least 30 days prior to surgery; the dose was adjusted to aim at a resting heart rate of 60–70 bpm. **[Q3: A, B, C, D]** The results showed that the incidence of the combined endpoint of cardiac death and myocardial infarction within 30 days of surgery was significantly lower in patients using bisoprolol compared to patients in the control group (combined endpoint 3.3% in the bisoprolol group vs. 34% in the control group). Based on the findings of these studies, beta-blocker use has been recommended by the ACC/AHA Guidelines on Perioperative Cardiovascular

Evaluation for Noncardiac Surgery in high-risk patients with a positive stress test as a level one recommendation [4].

3-Hydroxy-3-Methylglutaryl Coenzyme A Reductase Inhibitors (Statins)

Although perioperative use of beta-blockers has been associated with a significant reduction in cardiac mortality and morbidity, still some patients with multiple cardiac risk factors and positive stress test results may remain at considerable risk for perioperative cardiac mortality [9]. For these patients additional cardioprotective medication such as statin use may offer an important addition to preoperative risk reduction strategies. The association between statin use and possible reduction in perioperative cardiac complications may result from the favorable actions of statins on atherosclerosis and from their vascular properties other than those attributed to cholesterol lowering [10–12]. **[Q4: A, B, C]** These so-called pleiotropic effects of statins may attenuate coronary artery plaque inflammation and influence plaque stability in addition to antithrombogenic, antiproliferative and leukocyte-adhesion inhibiting effects [13–15]. All these effects of statins may stabilize unstable coronary artery plaques, thereby reducing myocardial ischemia and subsequent myocardial damage.

There are only a few studies that have evaluated the beneficial effects of perioperative statin use in reducing perioperative cardiac complications [16–18]. Poldermans et al. [16], using a case-control study design in 2816 patients who underwent major vascular surgery, showed that controls more often were statin users than cases, which resulted in a fourfold reduction in all-cause mortality within 30 days after surgery. This finding was consistent in subgroups of patients according to type of vascular surgery, cardiac risk factors and beta-blocker use. **[Q2: D]** Similar to these findings, Durazzo et al. [17] also reported a significantly reduced incidence of cardiovascular events within 6 months of vascular surgery in patients who were randomly assigned to atorvastatin compared with placebo (atorvastatin vs. placebo, 8.3% vs. 26.0%). Finally, the study results of Lindenauer et al. [18] indicated that statin use was associated with 28 percent relative risk reduction of in-hospital mortality compared to no statin use in 780,591 patients undergoing major noncardiac surgery. **[Q4: D]** The results of these studies are important indications of the possible beneficial effect of perioperative statin use. However, certain limitations such as the retrospective nature of the study of Poldermans et al. and Lindenauer et al., the relatively small sample size ($n = 100$ patients) of the study of Durazzo et al., and the lack of information about the optimal timing and duration of statin therapy warrant future clinical trials to confirm the effectiveness and safety of statin therapy in patients undergoing major noncardiac surgery. Initially, statin use was contraindicated in the perioperative period as it was thought that drug interactions might increase the incidence of myopathy and in combination with analgesics this might even remain asymptomatic. However, a recent study showed no increased incidence of myopathy among statin users [19]. Statin users undergoing vascular surgery at the Erasmus MC were screened for myopathy by measuring creatine kinase (CK) levels at regular intervals and checking for clinical symptoms. In 981 patients no relation was found between statin use and CK levels. Also, no patient experienced myopathy symptoms. Importantly, no deleterious effect of temporary statin interruption was observed. **[Q2: C and Q4: B, C]**

Preoperative cardiac risk evaluation may identify high-risk patients for whom the risk of perioperative cardiac complications without further coronary assessment and subsequent intervention could be too high. For these patients either percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass grafting (CABG) may be considered.

Percutaneous Revascularization

There have been several studies evaluating the clinical utility of PTCA in high-risk patients undergoing major noncardiac surgery including vascular surgery. In the studies of Elmore et al. [20] and Gottlieb et al. [21], retrospective data were collected of patients who underwent PTCA prior to surgery. These patients were referred for PTCA because of the need to relieve symptomatic angina or to treat myocardial ischemia identified by noninvasive testing. The findings of these studies indicated that the incidence of perioperative cardiac death and myocardial infarction was low, but the investigators in these studies failed to use a comparison group of patients with coronary artery disease not treated with PTCA. The apparent limitations of these studies prompted Posner et al. [22] to conduct their own investigation to compare adverse cardiac outcomes after noncardiac surgery in patients with prior PTCA, patients with non-revascularized coronary artery disease and normal controls. The results showed that patients treated with PTCA within 90 days of noncardiac surgery had a similar incidence of perioperative events to matched patients with coronary artery disease who had not been revascularized. **[Q5: A]** Those patients who underwent a PTCA procedure 90 days earlier then the day of noncardiac surgery had a lower risk of cardiac events than non-revascularized patients but not as low as normal controls. Furthermore, the effect of revascularization was limited to a reduction in the incidence of angina pectoris and congestive heart failure and there was no reduction in the incidence of death and nonfatal myocardial infarction. Indeed, the recent findings of the Coronary Artery Revascularization Prophylaxis (CARP) trial [23] also showed that coronary revascularization with PTCA or CABG prior to vascular surgery in high-risk cardiac stable patients did not provide short-term survival benefit or better long-term event-free survival rate. **[Q5: B, C, D]** The findings of the study indicated that patients undergoing coronary revascularization prior to vascular surgery had a 3.1 percent mortality rate within 30 days of vascular surgery compared to a 3.4 percent rate for those not having coronary revascularization ($p = 0.87$). Additionally, the rate of perioperative nonfatal myocardial infarction as detected by troponin elevation was also similar in coronary revascularization patients and patients not undergoing coronary revascularization (11.6% vs. 14.3%, $p = 0.37$). Furthermore, the results of the trial also indicated that coronary revascularization prior to vascular surgery was associated with delay or cancellation of the required vascular operation. Apart from these findings, it is also important to note that if a PTCA procedure and coronary stent placement are performed less than 6 weeks before major noncardiac surgery, the risk of perioperative coronary thrombosis or major bleeding complications may be substantially increased [24, 25]. Two separate small-scale studies reported an increased rate of serious bleeding complications if antithrombotic therapy was continued until the time of surgery, and in patients in whom antiplatelet drugs were interrupted one or two days before surgery an increased rate of fatal events was observed due to stent thrombosis [24, 25]. The risk of these complications persisted for 6 weeks after

coronary stent placement. Patients who underwent surgery more than 6 weeks after coronary stent placement experienced no adverse cardiac events. These observations indicate that if PTCA with stenting is planned in the weeks or months before noncardiac surgery then a delay of at least 6 weeks should occur before noncardiac surgery to allow for completion of the dual antiplatelet therapy and re-endothelialization of the stent.

Coronary Artery Bypass Grafting

The results of the largest retrospective study to date indicated that CABG had a protective effect prior to noncardiac surgery [26]. Data for 3368 patients analyzed from the Coronary Artery Surgery Study (CASS) registry showed that patients who underwent CABG before abdominal, vascular, thoracic, or head and neck surgery had a lower incidence of perioperative mortality (3.3% vs. 1.7%) and myocardial infarction (2.7% vs. 0.8%) compared with medically treated patients. The largest reduction in perioperative mortality was observed in patients with a history of advanced angina and in patients with multivessel coronary artery disease. In a more recent study, data analyzed from a random sample of Medicare beneficiaries showed that preoperative coronary revascularization was associated with a reduction in 1-year mortality for patients undergoing aortic surgery but showed no effect on mortality in those undergoing infrainguinal procedures [27]. Hassan et al. [28], using data from the Bypass Angioplasty Revascularization Investigation, showed there was no difference in the incidence of cardiac death and myocardial infarction between patients who underwent coronary angioplasty or CABG and subsequent noncardiac surgery (coronary angioplasty group, 1.6% vs. CABG group, 1.6%). **[Q5: A]** As mentioned above under 'Percutaneous revascularization', the recent findings of the CARP trial showed that high-risk patients randomized to coronary revascularization prior to vascular surgery had no better perioperative and long-term cardiac complication rates than medically treated patients. Therefore, in the light of these findings a decision to proceed with coronary angioplasty and selective revascularization before high-risk surgery should be made independent of the need for major noncardiac surgery [4].

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2. Abdominal Aortic Aneurysm

Jean-Pierre Becquemin and Alexandre d'Audiffret

A 59-year-old man presented with an abdominal aortic aneurysm (AAA) discovered on duplex scan examination of the abdomen. The AAA was 60-mm large and extended to the left common iliac artery. The patient was otherwise asymptomatic, with no abdominal or back pain. His medical history was significant for hypertension controlled by bitherapy, non-insulin-dependent diabetes diagnosed 5 years previously, claudication with a walking distance of 400 metres, and a smoking history of 40 packs/year. He had no history of myocardial infarction (MI) or angina pectoris.

He had a positive family history for an aneurysm. His father underwent surgery 20 years earlier for an abdominal aneurysm. He also has a brother who is 70 years old and a sister who is 55 years old with apparently no health problems

On examination, the patient was obese. No abdominal mass was palpated.

A computed tomography (CT) scan was performed (Figs 2.1 and 2.2).

Routine blood tests were normal except for serum creatinine level, which was 200 mg/ml. Electrocardiogram (ECG) was normal.

Question 1

The AAA of this patient was found during a routine screening. In which group(s) of population is duplex scan screening for AAA justified?

A. Patients with uncomplicated hypertension.

B. Patients with a family history of aneurysmal disease.

C. Patients with a smoking history.

D. Patients with peripheral vascular disease.

E. Obese patients with vascular risk factors

F. All men, starting at the age of 50 years.

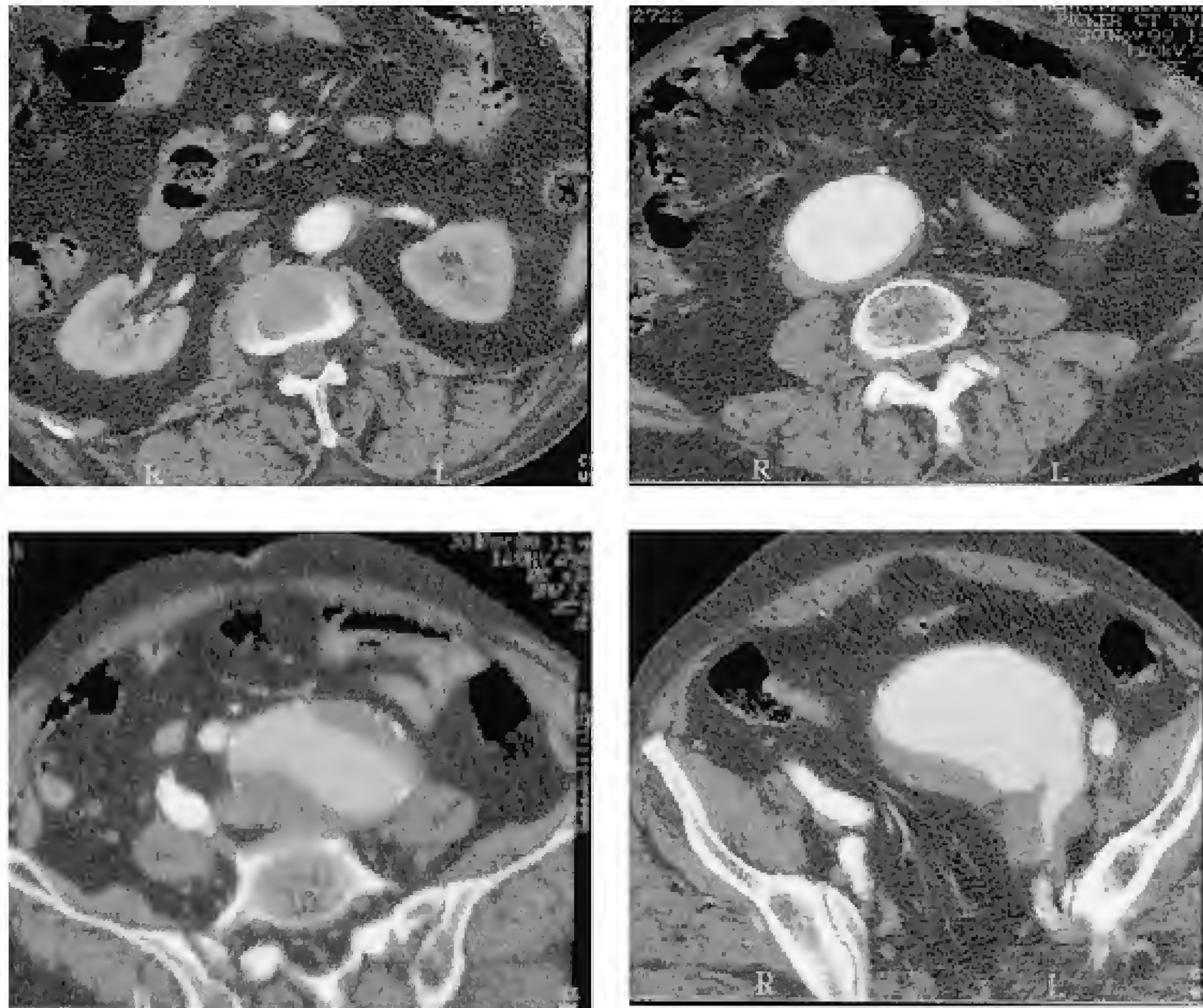


Fig. 2.1. CT scan demonstrating aortic, left common iliac, and left hypogastric aneurysms.



Fig. 2.2. Arteriography demonstrating aortic, left common iliac, and left hypogastric aneurysms (shadow of the external iliac indicated by arrow).

Question 2

With the imaging you have been provided with, is (are) there any reason(s) for performing an arteriogram?

A. No need, CT scan is sufficient.

B. Arteriogram is mandatory to define the surgical strategy.

C. Hypogastric aneurysm is a reason for performing an angiogram.

D. Angiography is required because of the suspicion of lower limb occlusive disease.

E. Serum creatinine level contraindicates angiography.

Question 3

To assess cardiac risk several tests can be performed. Which of the following explorations should be performed first?

A. None, ECG is sufficient.

B. Cardiac scintigraphy.

C. Cardiac echography.

D. Cardiac echography with dobutamine test.

E. Coronarography.

Question 4

Without treatment this patient is at risk of rupture. Which of the following factors have been proved to be associated with an increased risk of rupture?

A. Diameter >60 mm.

B. Association with a hypogastric aneurysm.

C. Diabetic patient.

D. Lower limb occlusive disease.

E. Smoking.

Question 5

If an operation were being considered, which of the following factors are associated with an increased postoperative mortality?

A. Diameter >60 mm.

B. Association with an hypogastric aneurysm.

C. Diabetic patient.

D. Renal insufficiency.

E. Smoking.

Question 6

With the current information obtained from the case description, what would you recommend to the patient?

A. Duplex scan surveillance every 3 months.

B. Aorto–bifemoral graft through a midline incision.

C. Aorto–bifemoral graft through a left retroperitoneal incision.

D. Aorto–bi-iliac graft through a left retroperitoneal incision.

E. Stent graft.

The patient underwent, via a left retroperitoneal approach, an aorto–right common iliac–left external iliac bypass with end-to-end anastomosis. The left common iliac artery was ligated at its origin. This was possible in this case because of the presence of a small proximal common iliac neck. The iliac and hypogastric aneurysms were opened, and back bleeding was controlled with an endoaneurysmal ligature. In addition, intraoperative cell-saver was used.

The left retroperitoneal approach was chosen because it permits an easier dissection of large left common iliac aneurysms, especially when associated with hypogastric aneurysms.

The patient's postoperative course was uneventful, and he was discharged on the tenth postoperative day.

Question 7

During open operation for AAA, cell-saver autotransfusion (CSA) can be used. Which of the following statements are correct?

A. It should be used routinely.

B. It should be reserved for when the expected blood loss is significant.

C. It should be substituted in all cases with preoperatively deposited autologous blood transfusion.

D. It presents fewer complications than unwashed cell autotransfusion.

One month later, the patient returned to the clinic with his 50-year-old brother who feared he could be suffering from a similar problem. He also stated that his father might have died from ruptured aneurysm.

Question 8

Does a genetic predisposition to AAA exist? Describe the pathogenesis of AAA.

Question 9

A duplex scan has been performed on the patient's bother, which detected a 40-mm abdominal aneurysm.

What recommendation(s) would you give this patient's brother?

A. Serial duplex studies at 3-monthly intervals, and intervention when the diameter reaches 5.5 cm.

B. Serial duplex studies at 6-monthly intervals, and intervention if the diameter reaches or exceeds 5 cm.

C. Serial duplex studies at 12-monthly intervals until the diameter reaches 4.5 cm, then every 6 months until the diameter reaches 5 cm, then every 3 months, and then intervention when the aneurysm reaches 5.5 cm.

D. Schedule the patient for surgery as he is a smoker and therefore his aneurysm will most likely require intervention.

Commentary

The question of the optimal format for population screening for AAA remains unanswered. Many studies have attempted to identify high-risk populations in order to reduce healthcare costs and maximise the yield. Simon et al. [1] have demonstrated a prevalence of AAA of 11 percent in male patients aged 60–75 years with a systolic blood pressure greater than 175 mm Hg. No patients with uncomplicated hypertension had an AAA. Claudication was the only cardiovascular complication associated independently with AAA (relative risk 5.8). Furthermore, preliminary results from the Aneurysm Detection and Management (ADAM) study revealed that smoking was the most important risk factor associated with AAA (odds ratio (OR) 5.57), followed by a positive family history (OR 1.95), age, height, coronary artery disease, atherosclerosis, high cholesterol level and hypertension [2]. Similar factors have been identified in the cardiovascular health study of the University of Pittsburgh [3]. Mass screening of the population, including all hypertensive patients, would not be cost-effective or beneficial. In conclusion, white men over the age of 60 years with significant hypertension should routinely be offered an abdominal ultrasound. **[Q1: B, C, D]**

As arteriography does not provide information on aneurysm size, the most important argument for routine arteriography as part of the preoperative evaluation of the patient with AAA is the detection of associated intra-abdominal vascular pathologies, especially renal artery stenosis or occlusion, presence of accessory renal arteries, coeliac and superior mesenteric stenosis or occlusion, and lower extremity disease.

According to several studies, the association of renal artery stenoses and AAA is found in 18–22% of cases [4]. Similarly, there is a prevalence of 25 percent for coeliac trunk stenoses and 6 percent for superior mesenteric stenoses in patients with AAA [5, 6]. Arteriography is associated with several complications related to its invasive nature and contrast load. Spiral CT provides high-quality images and

accurately identifies renal and visceral artery stenoses; however, the radiation and contrast requirements remain high. As a general rule, most vascular surgeons would agree on the selective use of arteriography and limit its use to patients with evidence of lower-extremity occlusive disease, suprarenal or juxtarenal aneurysms, and common or internal iliac artery involvement. Of note, when endovascular treatment is considered, an arteriogram with a graduated catheter is usually recommended for endograft sizing. However, in institutions where stent grafts are readily available on the shelf, intraoperative arteriogram is the first step of the procedure.

Moderate renal insufficiency is not an absolute contraindication to performing either an arteriogram or a stent graft. However, correct patient hydration and a small amount of contrast medium are necessary to avoid acute renal insufficiency. **[Q2: B,C]**

Routine coronary angiography in vascular patients has shown that 60% of them have severe coronary artery disease [7]. However, a large randomised study in patients with stable angina has clearly demonstrated that preoperative coronary bypass or angioplasty does not improve the postoperative and 5-year survival rate [8]. Beta-blockers, statins and antiplatelets have all contributed to the reduction of cardiac events following major vascular surgery [9]. Thus preoperative exploration can be restricted to patients with identified risk factors for severe coronary artery disease [10]. In the current case diabetes, renal insufficiency and claudication are three of these markers and preoperative cardiac screening should be undertaken. Dobutamine echo stress testing is probably the most reliable test [11]. Coronary angiography is indicated only when more than three parietal wall segments are ischaemic. **[Q3: D]**

The natural history of aneurysms and risk of rupture are better understood. A cohort study of patients who refused early operation [12] or who were considered to be inoperable has shown that the risk of rupture is significant for >5.5-cm aneurysms. A population-based study [13] has shown that diameter, FEV1 less than 1.5 l/min and smoking are strongly correlated with aneurysm growth and rupture. **[Q4: A, E]**

Analysis of factors predictive of mortality in patients submitted to open repair of AAA has shown that age, cardiac status, renal insufficiency and pulmonary status are strongly predictive of postoperative complications and deaths [14]. Difficult operations are also associated with an increased operative risk mostly related to the increased blood loss. Unilateral or bilateral hypogastric aneurysm increased the operative risk [14]. **[Q5: B, D]**

Surveillance was not recommended due to the aneurysm's size and the relatively young age of the patient.

This patient presented several factors which would make stent grafting a wise option. Unfortunately, the anatomy was not favourable. Huge left hypogastric aneurysms made a stent graft difficult with a risk of type II endoleak. Hypogastric embolisation if performed should have blocked the distal tributaries of the internal laic artery, which is a cause of sever buttock complications. Another restricting factor for stent graft is the creatinine level. Since CT scan with contrast medium is the gold standard for surveillance after stent grafting, renal function impairment is a serious risk in this patient.

Open surgery could have been performed with any of the proposed routes. However, median laparotomy was not the best choice due to the increased risk of postoperative abdominal wall dehiscence in this obese patient. Aorto–bifemoral bypass should be avoided to limit the risk of infection from the groin.

The left retroperitoneal approach was chosen because it permits an easier dissection of large left common iliac aneurysms, especially when associated with hypogastric aneurysms. **[Q6: D]**

Over the past three decades, with the appreciation of the risk of transfusion-related transmission of infectious diseases, a large body of research and instrumentation has emerged on autotransfusion. The current options are:

- preoperative deposit of autologous blood;
- intraoperative salvage and washing of red blood cells (cell-saver autotransfusion, CSA);
- intraoperative salvage of whole blood without washing.

Although both whole-blood autotransfusion (WBA) and CSA are currently in use, the magnitude of haemostatic and haemolytic disturbances, as well as the clinical side effects, after WBA compared with CSA are still under debate. While Ouriel et al. [15] showed the safety of WBA in 200 patients undergoing AAA repair, others have demonstrated a lower content of haemolytic degradation products and fewer coagulation disturbances after retransfusion of cell-saver blood [16]. Nevertheless, despite its widespread use, several studies have found that CSA is not cost-effective and should be limited to patients who have an expected blood loss of at least 1000 ml, which includes patients with large, complicated aneurysms [17, 18]. Finally, transfusion of pre-donated autologous blood is associated with some of the disadvantages of homologous transfusions, namely dilutional hypofibrinogenaemia, thrombocytopenia and hypothermia. **[Q7: B, D]**

The causes of AAA are numerous, and may include syphilis, aortic dissection, Ehlers–Danlos syndrome and cystic medial necrosis. However, more than 90 percent of all AAAs are associated with atherosclerosis and are classified as either atherosclerotic or degenerative aneurysms. The true aetiology of AAA is most likely multifactorial, including genetic predisposition, abnormality of the arterial wall proteolytic activity and connective tissue metabolism, and haemodynamic stresses.

The notion of genetic predisposition to AAA has been demonstrated in several reports of familial clustering as well as animal models. It is estimated that 15 percent of patients presenting with an AAA have a first-degree relative with the same condition. Male siblings are at higher risk, but current evidence also supports an autosomal dominant pattern of inheritance [19].

Multiple studies of the aortic aneurysmal wall have demonstrated an increased activity of metalloproteases, which have predominantly collagenolytic and elastinolytic activity, leading to fragmentation of the elastin network and replacement by a non-compliant collagen layer. In addition, deficiencies of tissue inhibitors of metalloproteases and alpha-1-antitrypsin have also been demonstrated [20].

Mechanical factors also play a significant role in the development of AAA. A strong association between hypertension and aneurysm expansion has been demonstrated. Also, the higher incidence of AAA in Second World War amputees places into play the reflective pressure stress [21]. **[Q8]**

The management and surveillance of small AAAs has been debated for many years. The UK Small Aneurysm Trial has attempted to shade some light on this subject [22]. The Trial Participants concluded that early surgical intervention did not offer any long-term survival advantages. Their recommendations, based on the trial methodology, were serial duplex every 6 months for aneurysms of size

4–4.9 cm, and every 3 months for aneurysms of size 5–5.5 cm. In another, larger analysis, the recommendations were yearly duplex for aneurysms measuring 4–4.5 cm on the initial scan [23]. **[Q9: B, C]** Indications and surveillance data for AAA remain confusing, however, and the answers to this question appear to represent common practice. One matter of concern is the common underestimation of the aneurysm size by duplex scan compared with CT scan, which may result in a high-risk surveillance period when the aneurysm diameter reaches 5 cm. Finally, according to the UK Small Aneurysm Trial, surgical treatment was unnecessary in only 25 percent of the patients, with 75 percent in the surveillance group requiring an intervention at some time during follow-up. Thus, the surveillance resulted only in a delayed intervention in 75 percent of cases. Chronic obstructive pulmonary disease (COPD) and continuation of smoking have been associated with aneurysm expansion, but the rate of expansion does not justify intervention on 4-cm aneurysms [24].

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3. Endoluminal Treatment of Infrarenal Abdominal Aortic Aneurysm

Hence J. M. Verhagen, Geoffrey H. White, Tom Daly and Theodossios Perdikides

A 78-year-old male was referred for investigation and management of an asymptomatic 6.2-cm diameter abdominal aortic aneurysm (AAA), which was diagnosed coincidentally during an abdominal ultrasound examination performed for investigation of prostatic symptoms.

The patient had a significant previous medical history, which included ischaemic heart disease, severe chronic obstructive lung disease, and several previous laparotomies. He had undergone coronary artery bypass graft (CABG) surgery a few years ago. On examination, the patient was haemodynamically stable with no abdominal complaints. There was an expansile pulsatile mass palpable in his obese abdomen, and all peripheral pulses were palpable.

Endoluminal repair of the AAA was considered, particularly in view of his high risk for open surgical repair.

Question 1

What is the optimal method of preoperative aneurysm assessment?

A. Abdominal colour flow duplex ultrasound.

B. Contrast-enhanced spiral computed tomography (CT) scan of the abdomen.

C. Abdominal colour duplex ultrasound and calibrated aortic angiography.

D. Contrast-enhanced spiral CT scan of the abdomen and calibrated aortic angiography.

Question 2

What is the mean annual risk of rupture of a 6.2-cm abdominal aortic aneurysm?

A. Less than 5 per cent.

B. Between 5 and 10 per cent.

C. Between 10 and 20 per cent.

D. Greater than 20 per cent.

Question 3

In anatomically similar aneurysms:

A. Open repair is a safer option for high-risk patients.

B. The benefits of endoluminal repair, in terms of reduced morbidity and mortality, only apply in high-risk patients.

C. The presence of chronic renal failure is an absolute contraindication to endoluminal repair.

D. Endoluminal repair results in a two-thirds reduction in 30-day operative morbidity and mortality compared to open aneurysm repair.

A contrast-enhanced spiral CT scan was obtained, which demonstrated that endovascular repair of the AAA was possible (Fig. 3.1). A calibrated aortic angiogram was performed to better define some of the anatomical features (Fig. 3.2). This showed an infrarenal aneurysm with a maximum diameter of 62 mm. The neck of the aneurysm was 19 mm in diameter and 22 mm in length. The distance from the lowest renal artery to the aortic bifurcation was 125 mm, and there was a further distance of 60 mm from the aortic bifurcation to the orifice of the internal iliac artery on each side. The neck of the aneurysm was noted to be quite angulated. The common iliac arteries were non-aneurysmal but severely tortuous, with angulations of more than 90° in their midsection. The minimum diameters of the external iliac and femoral arteries were 9 mm bilaterally. An endoluminal repair procedure was planned.

Question 4

Which anatomical features limit endoluminal repair?

A. Length and diameter of the aneurysm neck.

B. Length and diameter of the aneurysm.

C. Angle of the neck as well as the angle of the iliac arteries.

D. Tortuosity and diameter of the iliac arteries.

Question 5

For choosing a suitable endoluminal graft, one must:

A. Take the graft that resembles your measurements most closely.

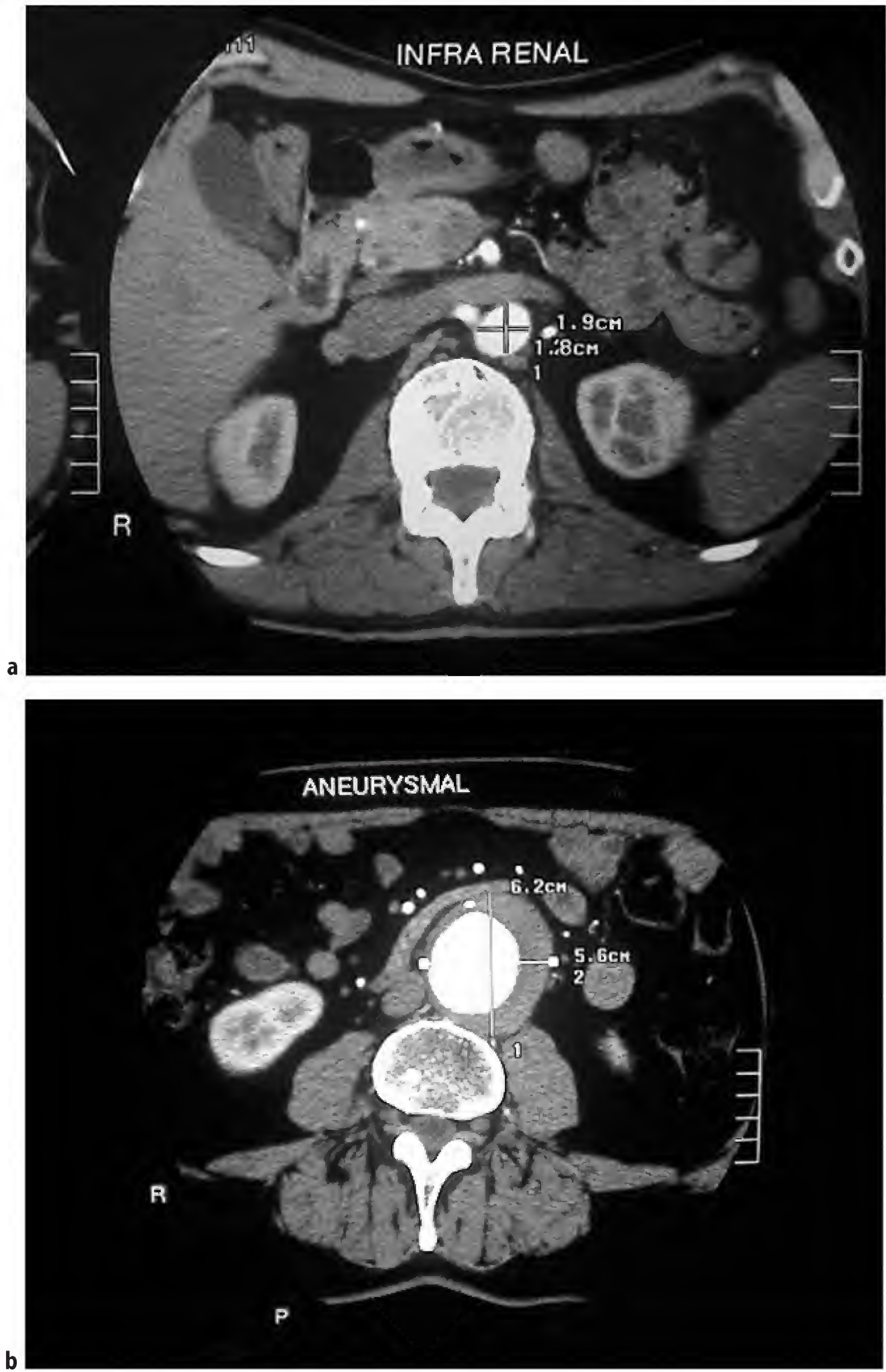


Fig. 3.1. (a,b)



Fig. 3.1. a Contrast-enhanced CT scan at the level of the neck of the aneurysm, immediately below the renal arteries. The neck has a maximum diameter of 19 mm on this image and has no irregular features, such as mural thrombus or atheromatous plaque. **b** Contrast-enhanced CT scan at the level of the sac of the aneurysm, showing a typical "target" appearance due to the presence of significant mural thrombus lining the aneurysm and contrast filling the flow channel. Maximum diameter 62 mm. **c** Contrast-enhanced CT scan at the level just below the aortic bifurcation, showing two iliac arteries of diameter 16–17 mm. **d** Three-dimensional reconstruction of a contrast-enhanced CT scan, showing angulation between the neck and the aneurysm of about 45°. The iliac arteries are tortuous, with an angulation of at least 90°.

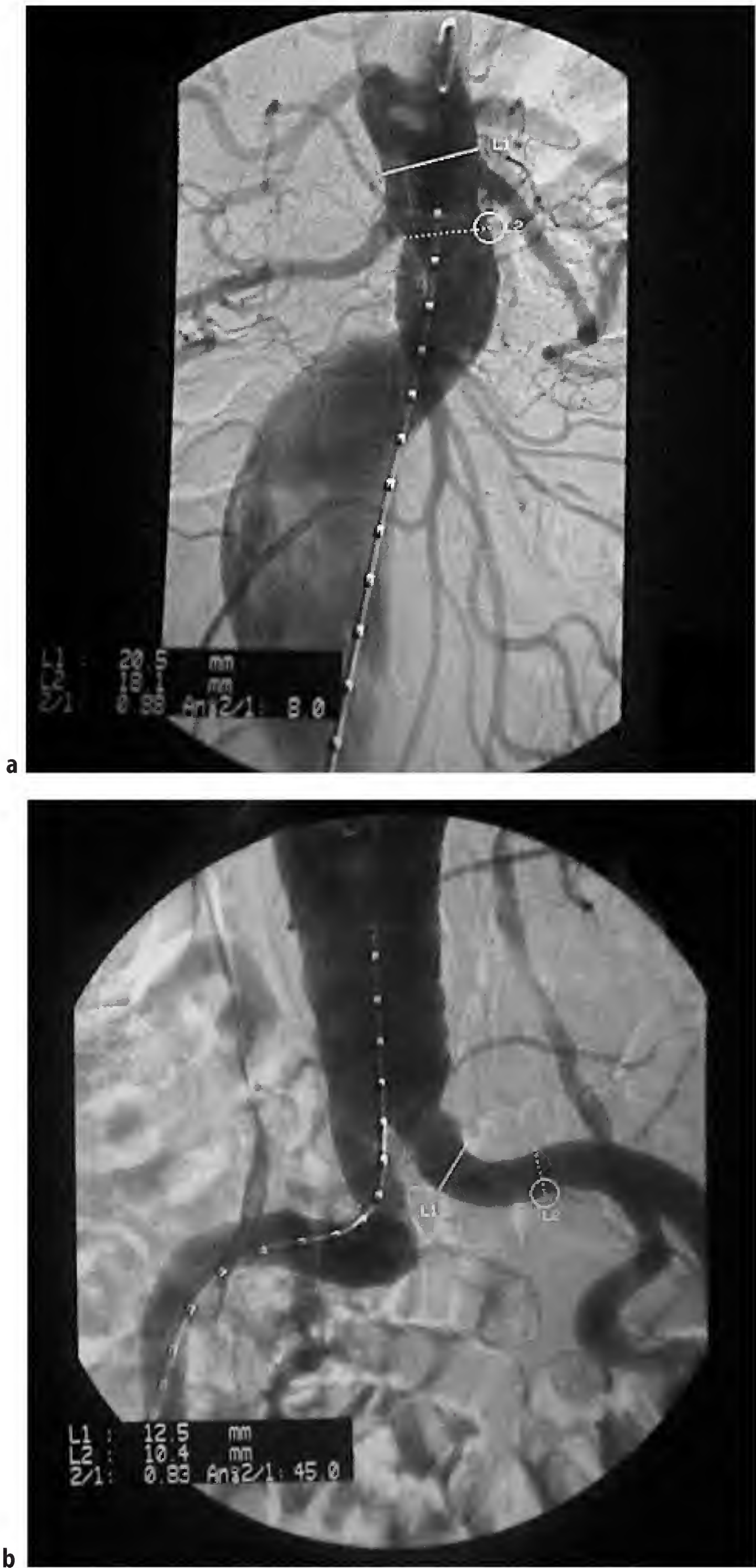


Fig. 3.2. a Calibrated aortic angiogram showing the aortic lumen, renal arteries and neck of the aneurysm. **b** Calibrated aortic angiogram showing the lumen of the aorta and both iliacs. Note that the diameter of the iliac arteries measures less on the angiogram than on the CT images.

B. Oversize all diameters by 10 per cent.

C. Oversize the proximal diameter by 20 per cent and the limb diameters by 10 per cent.

D. Undersize all diameters by 10 per cent and balloon-expand them to the proper size at the end of the procedure.

Due to the patient's severely impaired lung function and other risk factors, the procedure was performed under epidural anaesthesia. The abdomen and both groins were prepared into a sterile field. Common femoral arteries were surgically exposed, sheaths were inserted, and wires were put into place under fluoroscopy. Angiography was performed to mark exactly the position of the renal arteries (Fig. 3.3), and an endoluminal device was implanted successfully (Fig. 3.4).

Question 6

Whilst deploying the graft, the following need to be considered:

A. The orientation of the graft.

B. The location of any renal accessory renal arteries.

C. The location of the aortic bifurcation.

D. The location of the bifurcation of the common iliac artery.



Fig. 3.3. Early on-table angiogram to locate precisely the renal arteries during graft implant procedure. The arrow indicates the left renal artery. The device is ideally placed immediately below the orifice of the renal artery (note that some designs of endograft incorporate a bare stent that projects over the renal orifices). 📖



Fig. 3.4. Angiogram of a successfully deployed graft showing patent renal arteries, the endograft on the outside of the contrast-filled lumen, and the absence of an endoleak (flow of blood into the AAA sac). The blush near the proximal end of the graft (arrow) is gas inside the superimposed intestine.

Question 7

What are the more common complications of endoluminal aneurysm repair?

Question 8

Which of the following is the most important finding on follow-up imaging

A. No change to the lung field on chest X-ray.

B. The diameter of the aneurysm compared to the original diameter.

C. The absence of a type II endoleak previously visible on CT.

D. The position of the graft on abdominal X-ray.

Commentary

AAAs are generally a disease of elderly white males. In men, AAAs start to occur at the age of 50 years, reaching a peak incidence of about 350/100,000 person-years by the age of 80 years. The prevalence of AAAs of at least 3 mm in diameter in men over 65 years is 7.6 per cent. In women, AAAs tend to occur a few years later in life. The age-adjusted incidence is four to six times higher in men than in woman. Age,

gender and smoking are the risk factors with the largest impact on AAA prevalence [1].

Although ultrasonography is the method of choice for population screening or follow-up measurements in patients with known aneurysms, ultrasound imaging alone gives insufficient information for preoperative assessment for repair procedures. For open repair, most surgeons recommend a preoperative imaging study with (spiral) CT scanning, which provides accurate information regarding aneurysm size and its relationship to branch vessels, as well as any anatomic anomalies [2].

Preoperative imaging is even more important when endovascular treatment is considered, because patient selection and sizing of the endograft depend on it. With contrast-enhanced spiral CT, the dimensions of the proximal neck can be determined accurately and the presence of calcification or mural thrombus noted. Although the anatomy of the iliac arteries and accessory renal arteries can be demonstrated by spiral CT, in most medical centres calibrated aortography is also performed to allow accurate measurements. Anteroposterior and lateral views are required to demonstrate tortuosity in the neck of the aneurysm and the iliac arteries [3]. Three-dimensional reconstructions of contrast-enhanced CT scans are being utilised increasingly in order to get a more detailed perception of the actual anatomy. These reconstructions may become the standard method for accurate sizing of endografts in the near future. **[Q1: D]**

The decision of whether to treat an AAA remains a difficult process in which multiple factors play a role. One important factor is the risk of rupture, which, unfortunately, will always be an estimate, since large numbers of patients with AAAs have not been followed up without intervention. Based on currently available data from the UK Small Aneurysm Trial, the annual risk of rupture is less than 1 per cent when the diameter is 4–5.5 cm, although the validity of this risk estimation was compromised by the fact that many patients in this trial received surgery at a diameter less than 5.5 cm due to other factors. With increasing diameters, the annual rupture rates have been estimated to be as follows: 5–6 cm, 5–15%; 6–7 cm, 10–20%; 7–8 cm, 20–40%; <greater than>8 cm, 30–50% [4]. **[Q2: C]**

Recent trials have demonstrated a reduction in 30-day morbidity and mortality rate in patients undergoing endoluminal aortic aneurysm surgery compared to the traditional open approach [5, 6]. These well-constructed randomised trials showed a reduction of approximately 65 per cent in the incidence of moderate to major complications following endoluminal repair compared to open repair. Current information indicates that the benefit is likely to be greater for high-risk patients. The presence of renal failure is not an absolute contraindication to the endovascular approach as various precautions can be taken to protect the kidneys, such as intravenous hydration, antioxidant medications or temporary dialysis. The average contrast use for the procedure is 120–150 ml. **[Q3: C, D]**

Not all aneurysms are anatomically suitable for endovascular repair. In general, all endoluminal grafts need areas of reasonably healthy vessel wall proximally and distally to be able to seal off the aneurysm from the blood flow. Most important for suitability are the size and morphology of the proximal neck (the segment of aortic wall between the lowest renal artery and the beginning of the aneurysm). The neck should consist of relatively normal aorta over a length of at least 1.5 cm, and the diameter should not exceed 30 mm. Another limitation for endovascular repair is the maximum angulation in the neck (should not exceed 60°) and the iliac arteries (ideally less than 90°) [7]. **[Q4: A, C, D]**

After precise measurements from angiography and CT scanning, the optimal size of the graft can be chosen. To anchor the proximal part of the graft firmly onto the native aortic wall, a constant radial force of the graft is necessary. To achieve this, it is important to oversize the graft. Unfortunately, some recent reports suggest that there may be slow ongoing expansion of the neck of the aneurysm after endoluminal AAA repair [8]. This may favour a more aggressive oversizing of the proximal part of the graft, but this could in turn cause infolding of the graft fabric, which may lead to failure to seal the AAA sac ("endoleak"). Current recommendations are to oversize the proximal part of the graft by 10–20 per cent and the limb diameters by 10 per cent. **[Q5: C]**

Most current devices for endovascular repair procedures depend on self-expanding stents or wireforms for attachment to the aortic wall. Most grafts are configured so that there is a marking indicating the contralateral limb. Preoperative measuring assessment of size is confirmed intraoperatively to prevent problems in length of the limbs. After the graft has been implanted, a pigtail angiographic catheter is reintroduced, and a post-procedure digital subtraction angiogram is performed. The contrast run is examined closely for the presence of extravasation of contrast, suggesting an endoleak. In some grafts, the aneurysmal sac will fill temporarily with contrast due to porosity of the graft material. A retrograde angiogram through the femoral sheaths is used to visualise a seal at the distal end of the graft. **[Q6: A, B, C, D]**

Complications of endoluminal AAA repair have been divided into remote/systemic and local/vascular. The remote/systemic complications are similar to, but less frequent than, those occurring after open AAA repair. The local/vascular complications are more specific for the endoluminal repair (Table 3.1). **[Q7]**

The follow-up of endoluminal aneurysm repair patients remains important in determining the long-term success of aneurysm exclusion. General recommendations include a physical examination and abdominal X-ray (AXR) plus contrast-enhanced CT scan within 1 week, then 6, 12 and 18 months after operation, and then annually. At present there are a number of registries such as Cleveland Clinic, and a European collaboration (EUROSTAR) [9] indicating an annual mortality rate of more than 1 per cent related to abdominal aortic aneurysms after endovascular repair of AAA (EVAR). It appears that 15–25 per cent of deployed grafts will require some secondary intervention. The requirement for secondary intervention is greater for earlier generation grafts, compared with the newer devices. This appears

Table 3.1. Local/vascular complications after endoluminal repair 🕮

Injury to access arteries
Embolisation
Distal ischaemia
Renal failure
Endoleak
Type I (proximal or distal attachment zones)
Type II (lumbar or mesenteric collateral channels)
Type III (fabric tear or modular dislocation)
Type IV (porosity leak)
Endotension
Graft limb thrombosis
Groin wound infection
Conversion to open repair

to be independent of the length of time the grafts remain in place. There is, however, significant improvement in quality-of-life measurements in patients with EVAR compared with open repair up to at least 6 months [10]. The importance of the type II endoleaks is unclear. However, they have been known to reappear after a period of absence. The position and the integrity of the graft is easily identified on plain abdominal X-ray. Whilst the aneurysmal sac can vary over time with endovascular repair, an expanding aneurysmal sac in the presence or absence (endotension) of endoleaks warrants further investigation and treatment. **[Q8: B]**

Case Analysis Quiz

A number of imaging examples are shown in Figs 3.5–3.8. Analyse the anatomical suitability of each case for the possibility of endoluminal graft repair of the AAA. In particular, determine the favourable and unfavourable features shown.

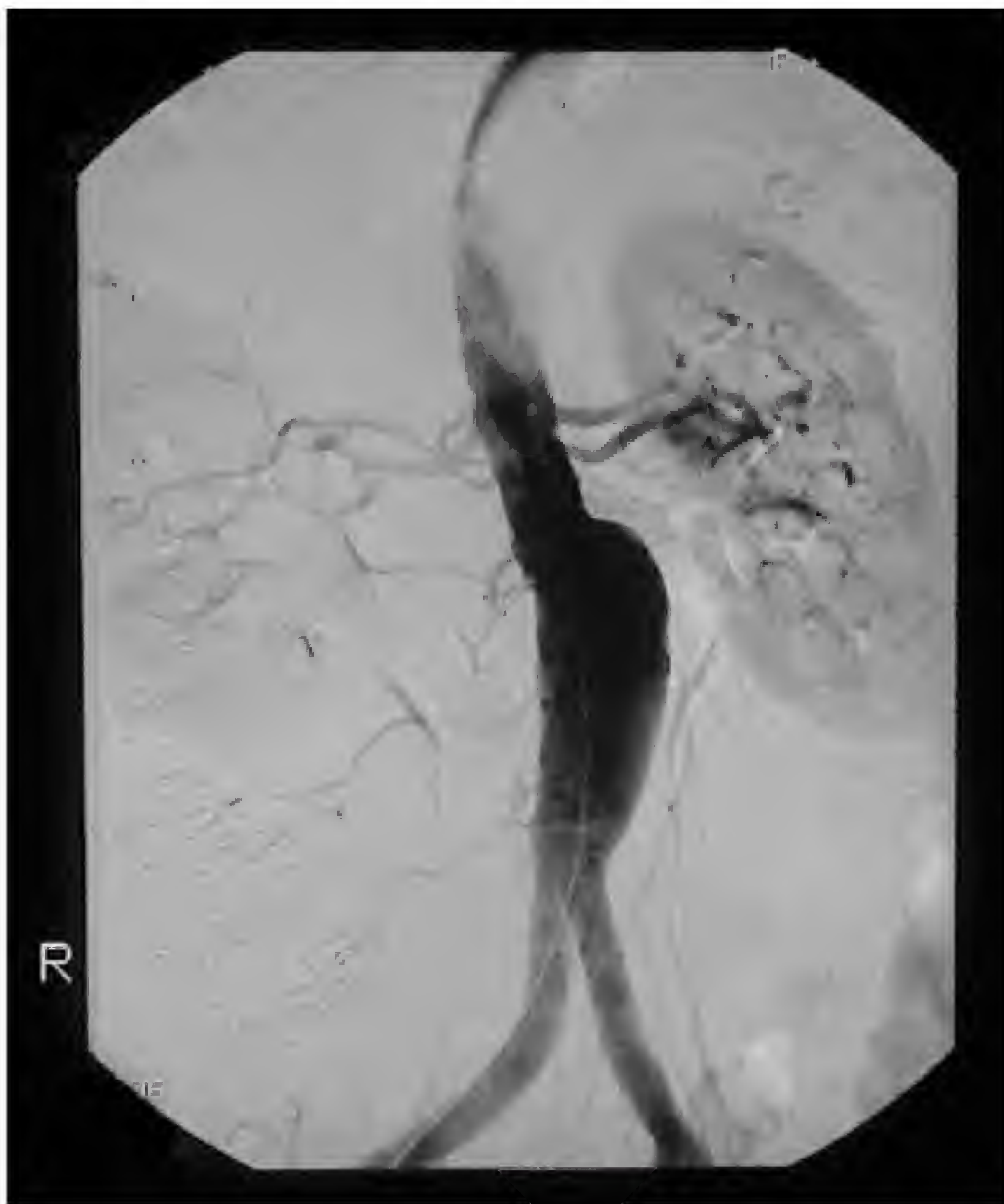


Fig. 3.5. Aortic angiogram showing a very favourable anatomy for endovascular repair: the neck is straight and long, without irregular features of the wall. In addition, the aneurysm sac is straight, and both iliac arteries are non-aneurysmal and relatively straight.



Fig. 3.6. In this case, the aortagram shows neck angulation of about 45°, and the neck has a reversed taper configuration. Both these features are considered to be unfavourable for endoluminal treatment because it is more difficult for the device to achieve complete seal and reliable fixation).

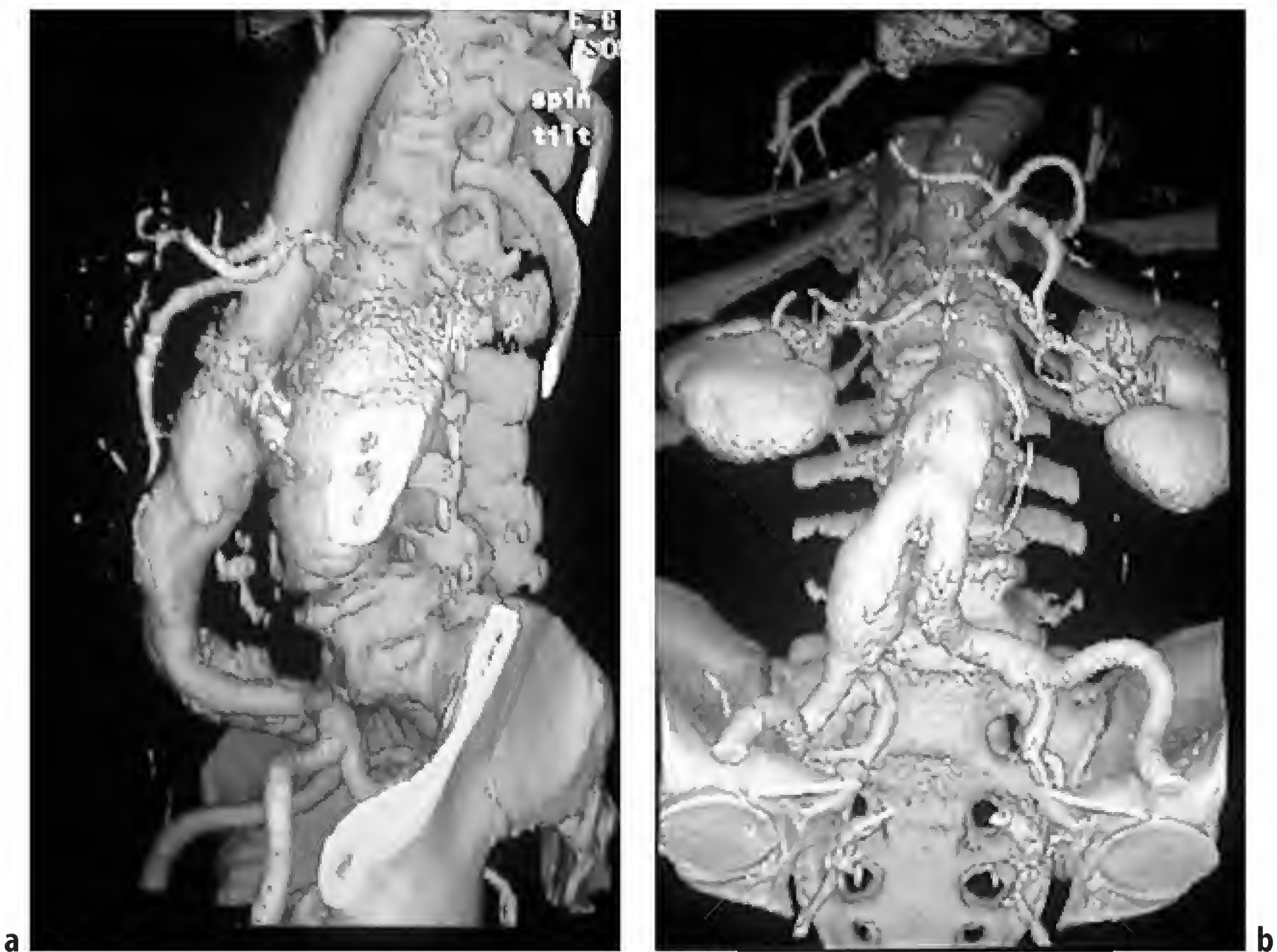


Fig. 3.7. Three-dimensional reconstruction of contrast-enhanced CT scan showing severe iliac angulation: **a** lateral view, **b** antero-inferior view. The iliac arteries show angulation in at least three different planes. The right common iliac artery is also aneurysmal. These features are unfavourable for access of the deployment sheath, and are associated with a higher risk of distal endoleak.



Fig. 3.8. Aortic angiogram showing a 90° angulation within the neck of the aneurysm. This is considered to be unsuitable for endovascular repair, since it is difficult to achieve satisfactory seal of the AAA sac or long-term attachment of the device.

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4. Ruptured Abdominal Aortic Aneurysm

Jeffrey S. Weiss and Bauer E. Sumpio

A 70-year-old white male presents to the emergency department with sudden onset of severe back pain. The pain is described as severe and constant without alleviating or aggravating symptoms. He has never had pain like this before. He denies chest pain, shortness of breath, or loss of consciousness. He denies any history of an abdominal aortic aneurysm. His past medical history is significant for hypertension, and chronic obstructive pulmonary disease that requires home oxygen therapy. He had bilateral inguinal herniorrhaphy some years ago, but has never had a laparotomy.

His vital signs yielded a pulse at 90 bpm and a blood pressure of 110/60 mm Hg. He is appropriately conversant and appears older than his stated age. He was without abdominal tenderness or masses and no bruits were heard; however, his belly was slightly obese and the examination was difficult. He has bilaterally palpable lower extremity pulses.

Question 1

What symptoms are considered the classic presenting triad for ruptured AAA?

A. Abdominal/back pain, shortness of breath, and a pulsatile mass.

B. Abdominal/back pain, syncope, and a pulsatile mass.

C. Abdominal/back pain, nausea, and syncope.

D. Abdominal/back pain, chest pain, and hematochezia.

The patient remained stable while the emergency department staff obtained laboratory results and cross-matched blood, and performed an electrocardiogram (ECG).

Question 2

If this patient is considered to have a ruptured AAA, which of the following factors does not adversely contribute to prognosis?

A. Diabetes.

B. Serum creatinine =1.8 mg/dl.

C. Age =75 years.

D. Preoperative blood pressure =80 mm Hg (systolic).

E. Syncope.

The patient's ECG shows normal sinus rhythm, the creatinine was 1.7 mg/dl, and the hematocrit was 32 percent. He remains hemodynamically stable. Your resident feels he is stable enough for a computed tomography (CT) scan (Fig. 4.1).

Question 3

Which of the following statements is true?

A. Patients with unknown AAA history and symptoms should undergo further diagnostic imaging if they are hemodynamically stable.

B. Symptomatic AAA should undergo emergency repair to prevent possible rupture.

C. Patients with an unknown AAA history must have diagnostic imaging confirmation of an AAA before proceeding to the operating theatre.



Fig. 4.1. Non-contrast CT scan of abdomen reveals an aortic aneurysm rupture in a left posterior location with extravasation into the retroperitoneum.



Fig. 4.2. Abdominal ultrasound with duplex color demonstrating rupture of aneurysm at the level of the left renal artery with a fluid collection in the left retroperitoneum.

D. An ECG demonstrating ischemic changes in a patient with epigastric pain, hypotension and tachycardia is the sine qua non for a myocardial infarction and any operation should be postponed.

E. CT scans are reserved for elective evaluation of AAA and have no place in the work-up of a symptomatic AAA.

Question 4

If an ultrasound (Fig. 4.2) was obtained instead of a CT scan, what statements could be made regarding this study?

A. Ultrasound is more reliable than CT scan for the diagnosis of ruptured AAA.

B. The location of the rupture is typical for most ruptured AAAs.

C. Ultrasound can be performed quickly at the bedside.

D. Ultrasound can be used to provide endograft measurements.

E. Ultrasound is best used in unstable patients to confirm the presence of a known AAA.

After the confirmation of ruptured AAA by radiology, the patient is taken immediately to the operating room.

Question 5

All of the following measures are indicated in the perioperative management of a ruptured AAA, except:

A. Surgical preparation and drape before induction.

B. Preoperative resuscitation to normal blood pressure.

C. Passive cooling of the patient.

D. Heparinization before cross-clamping.

E. Blood recuperation and autotransfusion devices.

The patient is prepared and draped, the anesthetic administered, and operation commenced. The medical student asks if this could be done via an endovascular approach.

Question 6

Currently, what are the contraindications for endovascular repair of ruptured AAAs?

A. Infrarenal neck diameter >30 mm.

B. Infrarenal neck length <10 mm.

C. Systolic blood pressure <100 mm Hg.

D. Endograft or "endograft team" not available.

E. Thrombus present at infrarenal neck.

The patient was determined to have too large a neck diameter for an endovascular stent, so you decide to proceed with an open repair. After induction, the patient's blood pressure falls to a systolic of 60 mm Hg. A supraceliac clamp is quickly placed and the aneurysm exposed. The rupture was contained to the retroperitoneum, but is rather large. The supraceliac clamp is moved to an infrarenal position after about 10 minutes. Anesthesia quickly catches up and his systolic blood pressure rises to 100 mm Hg. The inferior mesenteric artery was not patent and the iliac arteries were without aneurysms, allowing a Dacron tube graft to be placed. The clamp is slowly removed and he remains hemodynamically stable. The bowel appears well perfused and distal pulses are palpable before closure. Postoperatively, the patient recovers in the surgical intensive care unit.

Question 7

The most common complication following repair of ruptured AAAs is?

A. Aortoenteric fistula.

B. Bowel ischemia.

C. Myocardial ischemia.

D. Atheroemboli.

E. Acute renal failure.

He is noted to have a creatinine that rises to 4.7 mg/dl 2 days after operation and his urine output falls to less than 100 ml/day. He is eventually placed on intermittent hemodialysis because of volume overload. Over the next 2 weeks he is weaned off the ventilator, his urine output slowly increases, and his creatinine levels stabilizes at 2.0 mg/dl. He is discharged to a convalescence facility 19 days after operation.

Commentary

The optimal treatment of ruptured abdominal aortic aneurysm (rAAA) is prevention; unfortunately close to 70 percent of presenting patients have no prior diagnosis [1]. The overall mortality rates for rAAA are 80–90 percent with operative mortality around 50 percent [2–4]. Although more than three-quarters of patients with an rAAA report either abdominal or back pain, they can present with a myriad of symptoms and signs that are both broad and inconsistently present [5]. The triad of hypotension, abdominal pain, and a pulsatile mass **[Q1: B]** are found together in only half of cases [6]. A great deal of effort has been applied to identifying perioperative risk factors for patients who have a decreased survival advantage. Preoperative risk factors include: age <75–76 years, hypotension =80–95 mm Hg, creatinine =1.8–1.9 mg/dl, loss of consciousness, ECG ischemia or dysrhythmia, CHF, hemoglobin <9 g/dl, base deficit >8, and free rupture [7–10]. **[Q2: A]** Intraoperative risk factors include: blood loss >2–3.5 liters, duration of surgery >200 min, aortic cross-clamp time >47 min, lack of autotransfusion devices, bifurcated grafts, and technical complications (i.e., left renal vein injury) [11–13]. Postoperative risk factors include renal failure, coagulopathy, and cardiac complications. Hardman et al. [10] found that possession of three or more preoperative risk factors correlated with 100 percent mortality. Currently, no recommendation exists to withhold surgery for patients with any or all of these risk factors; this decision is made on a case-by-case basis, making risk factor analysis useful mostly from the standpoint of guiding patient decisions on surgery and family discussions on prognosis.

Patients who present with symptoms of a rAAA can be divided into two groups based on whether or not they have a known AAA (Fig. 4.3) [14]. Unstable patients with known AAAs present the least diagnostic challenge as they belong in the operating room. In contrast, the unstable patient without known AAA can be the hardest to evaluate. If an rAAA is suspected, this patient needs to be assessed expeditiously with an ECG as myocardial infarction can often mimic these symptoms. If cardiogenic shock is clinically apparent, resuscitation should override emergent surgery; however, cardiac ischemia secondary to hypovolemic shock from a rupture needs both rapid resuscitation and emergent surgery as the underlying cause of shock is the rupture and not the heart. Patients without hemodynamic instability allow the examiner the time to proceed with radiological confirmation [15]. **[Q3: A]** Ultrasound is fast and convenient as it allows an examination while resuscitation is taking place at the bedside. The sensitivity is as high as 100 percent for detecting an AAA, but it is inaccurate on diagnosing rupture (49 percent) [12,16]. This study is ideal on hemodynamically stable patients without known AAA, minimal operative risk factors, and symptoms or signs suggestive of rupture. **[Q4: B, C]** In this case, the mere presence of an AAA would warrant surgery without delay. CT scans are more difficult to obtain and place the patient at some increased risk because of time delay and interruption of resuscitation. They are clearly only indicated for patients who

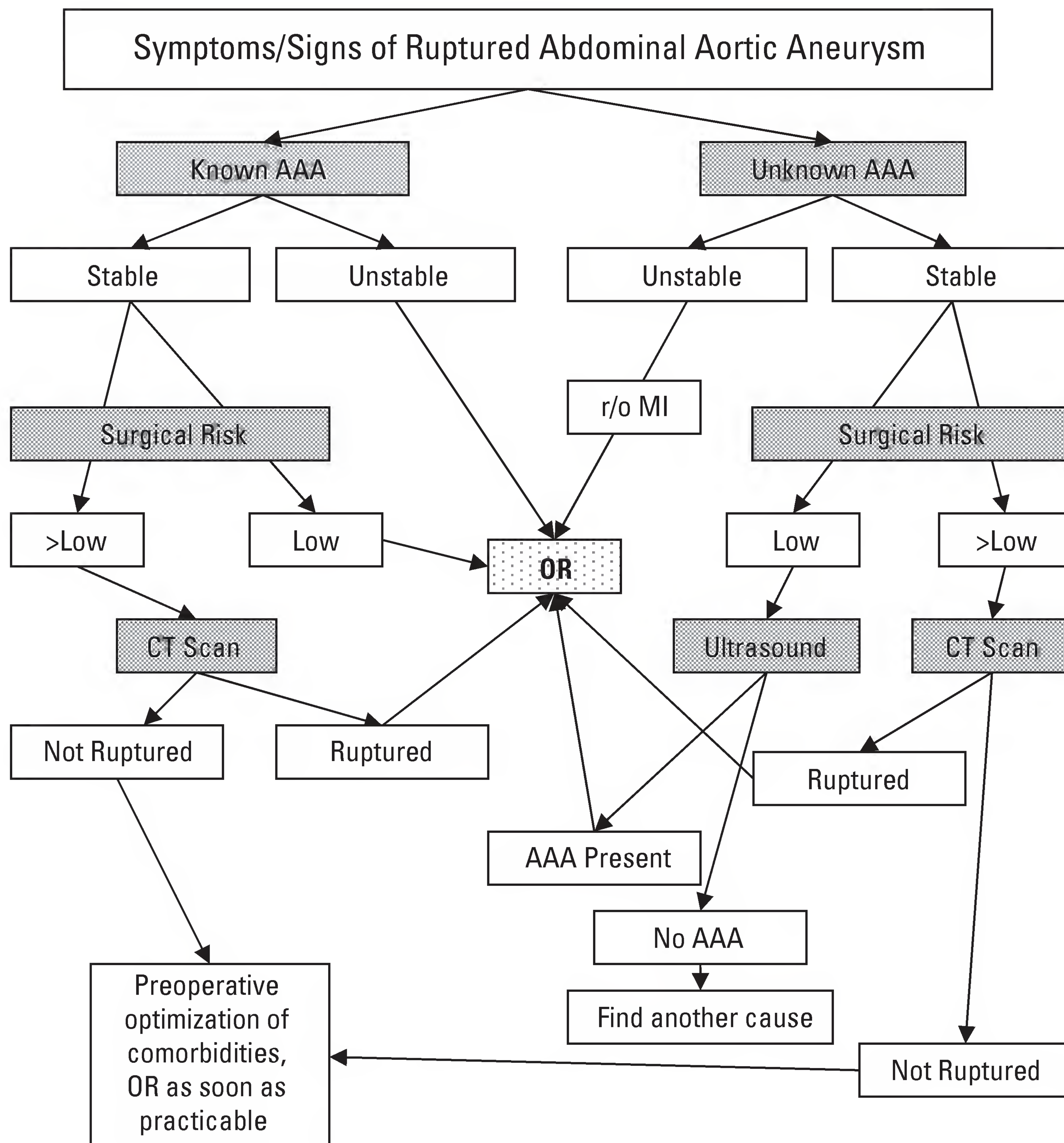


Fig. 4.3. Algorithm for suspected rAAA.

are stable and offer the advantage of being able to diagnosis rupture. The groups of patients most likely to benefit from CT scan are those with significant comorbidities where delay could allow preoperative optimization [17]. The sensitivity and specificity of CT scan for diagnosing rupture is quoted to be as high as 94 percent and 95 percent, respectively [15].

Once the decision to operate has been made, several preoperative measures should be undertaken. A natural instinct is to bolus intravenous (IV) fluid in an attempt to normalize the blood pressure; this should be avoided. Instead, adopting a *permissive hypotensive* strategy will allow the patient's own physiologic response to minimize blood loss [18]. Although there are times when fluids are necessary, this strategy can be effective in preventing accelerated blood loss until the aorta is clamped or occluded. Every effort should be made to keep the patient warm with blankets, raising the operating room temperature, and utilizing warmed IV fluids

and blood products [8]. The patient should be prepared and draped before induction as the loss of sympathetic tone with anesthesia may cause a marginally compensated patient to collapse.

A midline laparotomy provides the quickest route of entry and best exposure in most cases. A low threshold to obtain supraceliac control will prevent inadvertent venous injury, especially in cases with large retroperitoneal hematomas. This control is obtained by incising the gastrohepatic ligament and diaphragmatic crura, and then bluntly dissecting the periaortic tissue; a preoperative nasogastric tube can aid in identification of the laterally positioned esophagus. A clamp or manual pressure is applied to the supraceliac aorta. The transverse colon is reflected cephalad and the small bowel eviscerated. The supraceliac control can then be moved to the infrarenal neck after it is carefully dissected out. Systemic heparinization is avoided and heparinized saline (10 units/ml) is used locally down both iliacs before balloon occlusion. The use of intraoperative blood recuperation and autotransfusion devices is crucial in minimizing postoperative mortality by limiting homologous blood transfusions [13]. The use of a tube graft, typically knitted Dacron or PTFE, will shorten operative times and restore flow sooner than a bifurcated graft; this may necessitate leaving aneurysmal iliac arteries alone [14]. **[Q5: B, C, D]** After completion of grafting, bowel and lower extremity perfusion are assessed, usually by inspection and Doppler probe. The aneurysm sac is closed around the graft in an attempt to prevent later aortoenteric fistulas. Depending on the size of retroperitoneal hematoma and degree of resuscitation, the abdomen may not close easily. In these cases, it is best to perform a temporary closure with plans to return to the operating room for washout and definitive closure at a later, more stable time.

The dismal mortality following open repair of rAAA and the expansion of endovascular techniques has prompted recent exploration into application of stent grafts for primary therapy. Patient candidacy for an endovascular repair of AAA (EVAR) is the first hurdle when considering this approach. Measurements to determine this are typically done by CT angiography, although the Montefiore group have been successful utilizing digital subtraction angiography in two views [19]. The concern of sending a potentially unstable patient with known or suspected ruptured AAA to the CT scanner was recently addressed by Lloyd et al. [20] from Leicester; they found that 87.5 percent of patients survived longer than 2 hours after admission, with 92 percent of these patients having systolic blood pressures greater than 80 mm Hg. Ruptured or symptomatic AAAs are found to have larger infrarenal neck diameters and smaller neck lengths [21]. Despite these morphological differences, several reports have found amazingly high feasibility rates for EVAR, ranging from 46 percent to 80 percent [22, 23]. Dimensional requirements for endografts are constantly shifting as new devices improve the field, but currently an infrarenal neck =10 mm and a diameter =30 mm are needed [24]. **[Q6: A, B, D]** The next hurdle is availability of an endograft team and the graft itself. The importance of a knowledgeable and experienced team cannot be overstated as any program without this is destined for failure. A variety of grafts are being utilized, with favor towards a modular aorto-uniiliac device; this set-up decreases the need for large inventories [23, 24]. The Montefiore group have developed an aorto-unifemoral graft which they use in conjunction with a crossover femoral-femoral graft [19]. Surprisingly few patients are rejected for EVAR secondary to unfavorable hemodynamics. Supraceliac balloon occlusion via a brachial or femoral route under fluoroscopic guidance can allow proximal aortic control under local anesthesia; a technique being utilized by some for control prior to laparotomy in open cases [25].

Prospective randomized studies are underway to examine the morbidity and mortality rates of EVAR with respect to open repair, but preliminary nonrandomized results are already favoring this approach [19, 24, 26].

The most common complication of rAAA repair is renal failure, followed by ileus, sepsis, myocardial infarction, respiratory failure, bleeding, and bowel ischemia [1, 11]. **[Q7: E]** Postoperative renal failure has been found by several authors to correlate with mortality [1, 11]. Minimizing suprarenal clamp time and use of mannitol before cross-clamping the aorta to initiate brisk diuresis may limit renal damage. The inflammatory mediators and cytokines released from the shock state, visceral hypoperfusion, and massive transfusions associated with open repair can lead to multi-organ system failure; the avoidance of supraceliac clamping and lower blood loss are some of the potential advantages of the EVAR approach. But EVAR has its own unique complications which include endoleaks, graft malfunction, and groin wound issues.

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5. Thoracoabdominal Aortic Aneurysm

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Julius H. Jacobson II

A 72-year-old white male presented to his primary-care physician with a history of left chest pain for the past month. The pain was dull and constant, and radiated to the back, medial to the scapula. He denied new cough or worsening shortness of breath. He had no recent weight loss, and his appetite was good. He has a history of hypertension that was currently controlled medically. He had a smoking history of 60 packs a year. In addition, he suffered a myocardial infarction (MI) 5 years ago. The patient denied any history of claudication, transient ischaemic attacks or stroke. He had undergone surgery in the past for bilateral inguinal hernias, and he underwent cardiac catheterisation after his MI.

On physical examination, the patient was thin but did not appear malnourished. Vital signs were respiratory rate 18/min, heart rate 72 bpm, blood pressure 140/80 mm Hg and temperature 36.8°C. His head and neck examination was remarkable only for bilateral carotid bruits. Cardiac examination revealed a regular rate and rhythm without murmurs. Abdominal examination revealed no bruits and an infrarenal aortic diameter of approximately 2.5 cm by palpation. His femoral and popliteal pulses were normal. Posterior tibial pulses were 1+ bilaterally, and dorsalis pedis were detectable only by Doppler. Routine blood work was unremarkable, and an electrocardiogram (ECG) revealed changes consistent with an old inferior wall MI and left ventricular (LV) hypertrophy. Chest X-ray (Fig. 5.1) was remarkable for a tortuous aorta, which had calcification within the wall and appeared dilated. There were no pleural effusions, there was some flattening of both hemidiaphragms, and bony structures were normal. Lung fields were clear of masses or consolidation.

Question 1

Which of the following is the single most likely diagnosis causing this man's pain?

A. Acute MI.

B. Acute aortic dissection.

C. Thoracic aortic aneurysm.

D. Lung cancer.

E. Pneumonia.



Fig. 5.1. Chest X-ray demonstrating tortuous and dilated descending thoracic aorta suggestive of a thoracoabdominal aorta.

Question 2

Which of the following studies should be performed in this patient in order to plan therapy?

A. Aortography.

B. Computed tomography (CT) scan of chest.

C. Carotid duplex studies.

D. Cardiac stress test.

E. Arterial blood gas (ABG) analysis.

The CT scans of the chest and aortagram shown in Figs 5.2 and 5.3 were obtained. Findings were consistent with a thoracoabdominal aneurysm without concomitant dissection of the aorta. There was no evidence for acute leak or rupture, and the maximal diameter of the thoracic aorta was 7.3 cm.

Question 3

Briefly describe the Crawford classification system for thoracoabdominal aortic aneurysms TAAs).



Fig. 5.2. CT scan demonstrating aneurysmal dilation of the descending thoracic aorta.

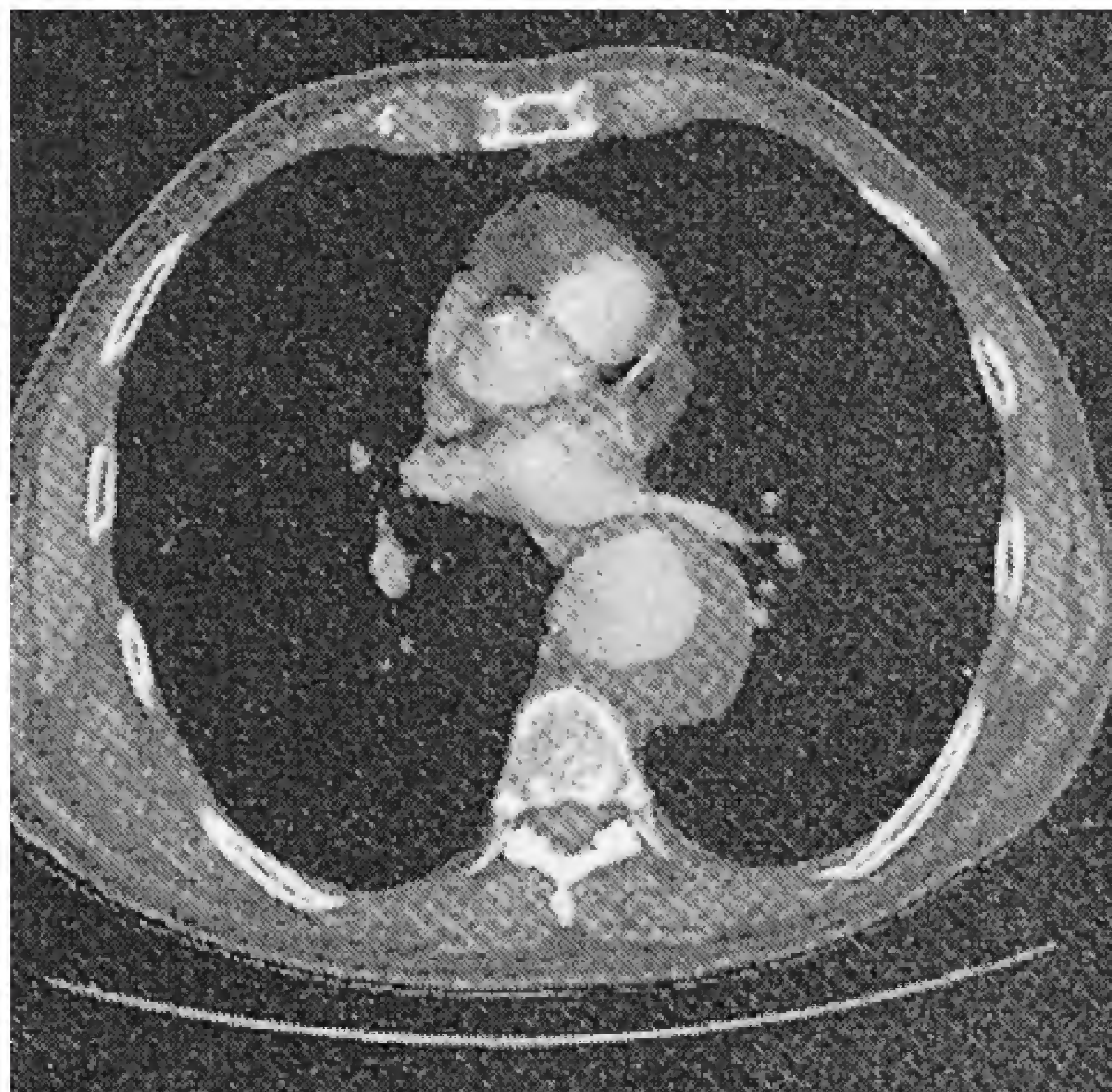


Fig. 5.3. Aortagram of patient in Fig. 5.2 showing tortuosity of aneurysmal aorta. Note the disparity between lumen size and aortic diameter, indicating a significant amount of mural thrombus.

The patient underwent a cardiac stress test, which was normal. Carotid duplex studies revealed bilateral stenoses of less than 50 per cent. ABG analysis showed pH 7.38, pCO_2 42 and pO_2 76 on room air.

Question 4

Which of the following management schemes seems most reasonable for this patient?

A. Observation with annual follow-up chest CT.

B. Repair of thoracoabdominal aneurysm after bilateral carotid endarterectomies.

C. Cardiac catheterisation followed by repair of TAA.

D. Elective repair of TAA.

The patient is scheduled for elective repair of his TAA. He expresses concern about the possibility of complications from the surgery. You explain to him the most likely complications related to this surgery.

Question 5

List the four most common complications following TAA repair.

The patient seems most concerned about the risk of postoperative paralysis. You explain to him that there are things you can do to decrease his risk of suffering this complication, although nothing can eliminate the risk.

Question 6

List four technical modifications that may be beneficial in the prevention of spinal cord dysfunction following TAA repair.

The patient undergoes repair of TAA and tolerates the procedure well. Postoperatively, the chest tubes are draining 100–150 cm^3 blood/hour for the first 3 h. In addition, urine output is steady at 500 cm^3/h. The patient has transient drops in blood pressure to a systolic blood pressure in the 70s, with central venous pressure dropping to 5 mm Hg.

Question 7

(a) Describe the initial work-up and potential correction of the bleeding problem described above in order to prevent a return to the operating room. (b) What fluid resuscitation approach should be taken to stabilise this patient's haemodynamic status?

The patient's temperature is 34.6°C, international normalised ration (INR) is 1.7 and prothrombin time (PTT) is 50 s (control, 34 s). Platelet count is 33,000. After

infusion of warm fluids, the use of a warming blanket, and platelet and fresh frozen plasma (FFP) transfusions, the parameters return to normal and the drainage from the chest tubes decreases to about 10–20 cm^3/h. On the second postoperative day, the patient is noted to have loss of motor function in his lower extremities.

Question 8

What therapeutic intervention may, if carried out in a timely fashion, restore this patient's neurological function partially or fully?

Following appropriate intervention, the patient's neurological function returns to normal. The patient's recovery is otherwise uneventful, and he is discharged on postoperative day 8 with clean incisions, intact neurological status and adequate analgesia.

Question 9

What is this man's approximate predicted 5-year survival?

Commentary

TAAs are less common than infrarenal abdominal aortic aneurysms. One population-based study suggested an incidence of 5.9 TAAs per 100,000 person-years [1]. Although TAAs are more common in males, the male : female ratio of 1.1–2.1 : 1 is not as weighted as the ratio of abdominal aortic aneurysm (AAA). The aetiology of TAAs is related to atherosclerotic medial degenerative disease (82 per cent) and aortic dissection (17 per cent) in most cases [2]. About 45 per cent of TAAs are asymptomatic and detected during work-up of other systems, usually on chest X-ray or cardiac echo exam. Patients tend to be older than AAA patients and therefore may have more severe comorbidities. When present, symptoms are usually pain related to compression of adjacent structures by the aneurysm or cough from compression/erosion of airways. Fistulisation into the bronchial tree can lead to massive haemoptysis, while erosion into the oesophagus can result in upper-gastrointestinal bleeding. Acute, severe pain may reflect leak, acute expansion or dissection of the aneurysm and may require more urgent evaluation and treatment. The risk factors associated with TAA are smoking, hypertension, coronary artery disease, chronic obstructive pulmonary disease, and disease in other vascular beds. Syphilitic aneurysms are a rare cause of TAA in this era and usually involve the ascending aorta. Other causes of vague chest and back pain in a patient such as this include myocardial ischaemia, pulmonary neoplasm, acute dissection, pneumonia, and bony metastases. **[Q1: C]** The clinical and X-ray findings in this particular case argue against these other possibilities.

The work-up of patients with TAA requires assessment of the aneurysm extent and size, as well as of the condition of the remaining aorta. **[Q2: A, B, C, D, E]** Before any studies are carried out, a thorough history and physical examination, including vascular assessment, are needed. Currently, aortography remains an important tool

for defining the extent of TAA as well as the status of aortic branches, but CT exam is the most useful diagnostic test. Helical CT scanning and CT angiography are rapidly becoming more precise in terms of delineating extent of aneurysm, as well as diameter, presence of dissection, presence of leak, and involvement of aortic branches. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) continue to improve and offer benefits over CT such as lack of radiation and non-nephrotoxic contrast agents. MRA has not yet achieved the resolution of conventional angiography, and its use is contraindicated in unstable patients. Transoesophageal echocardiography can assess the status of the aortic valve as well as cardiac function. Significant aortic insufficiency is a contraindication to thoracic aortic cross-clamping unless a shunt or pump is used to bypass the left heart. In addition to assessment of the aneurysm, the high incidence of comorbidities in this patient population mandates thorough evaluation of cardiac as well as pulmonary reserve. Preoperative studies should include EKG and cardiac stress testing. Further work-up will be dictated by the presence of positive findings. Screening chest X-ray and preoperative ABG will provide information regarding pulmonary status. Formal pulmonary function tests should be reserved for those patients with evidence of significant pulmonary compromise. Since the risk factors for TAA are the same as those for atherosclerotic disease, a careful history and physical will dictate whether there is a need to work up disease in other beds (carotid, mesenteric, renal, lower extremity). Carotid duplex studies may be done routinely preoperatively and significant stenoses treated before TAA repair. The status of the patient's clotting system must be determined and optimised if necessary. In the absence of indications to carry out other operations first, this patient with a TAA of >6 cm should undergo elective repair of his aneurysm. **[Q4: D]** Observation with follow-up imaging studies is dangerous and puts the patient at risk of death due to aneurysm rupture.

The Crawford classification **[Q3]** is used to characterise TAAs (Fig. 5.4) [3]. According to this system, aneurysms beginning just distal to the left subclavian artery and involving the aorta up to but not below the renals are termed type I. Type II begin aneurysms begin just beyond the left subclavian and continue into the infrarenal aorta. Type III aneurysms involve the distal half of the thoracic aorta and varying extents of the abdominal aorta, while type IV refers to those aneurysms involving the entire abdominal aorta, up to the diaphragm and including the visceral segments. This classification scheme has been useful for predicting morbidity and mortality following repair of TAAs. In the case of non-dissecting TAA, the four types occur with approximately equal frequency.

The natural history of TAAs is related to size and growth rate. Understanding the behaviour of these lesions is of crucial importance when determining treatment. Crawford's series of 94 TAAs followed for 25 years demonstrated 2-year survival of 24 per cent, with about half of deaths due to rupture [4]. This series included dissected as well as non-dissected aneurysms. A more recent series of non-dissected TAAs revealed rupture rates of 12 per cent at 2 years and 32 per cent at 4 years; for aneurysms greater than 5 cm in diameter, rupture rates increased to 18 per cent at 2 years [5]. Rupture is very uncommon in aneurysms measuring less than 5 cm in diameter. Another risk factor for rupture seems to be expansion rate, with aneurysms growing more than 5 mm in 6 months at higher risk than those growing more slowly. Survival in nonoperated patients was 52 per cent at 2 years and 17 per cent at 5 years. Patients who underwent repair of TAA had a 5-year survival of 50 per cent. Another series revealed 61 per cent 5-year

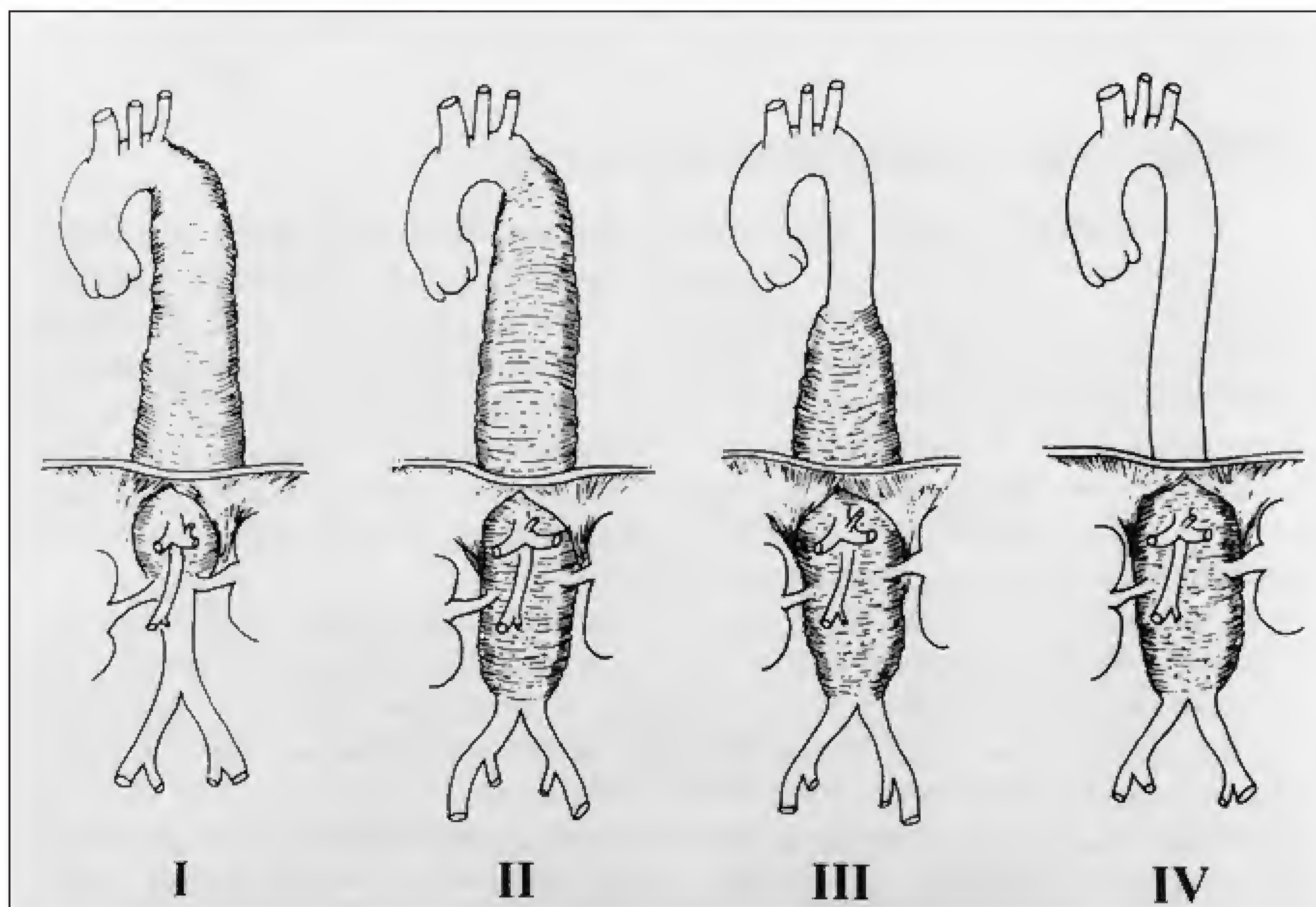


Fig. 5.4. Crawford classification of thoracoabdominal aortic aneurysms, types I–IV.

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survival following TAA repair. Survival decreased to 50 per cent for patients with dissecting TAA [6]. **[Q9]**

Operative repair is usually through a left thoracotomy with a paramedian abdominal extension, depending on the distal extent of the aneurysm. A retroperitoneal approach to the abdominal segment is used. The distal extent of the aneurysm determines which intercostal space will be used for a thoracotomy. The incision is in the fourth or fifth intercostal space for type I or high type II TAA, while an incision in the seventh, eighth or ninth space is appropriate for types III or IV [7]. Careful identification and reimplantation of visceral vessels is important, as is reattachment of intercostal arteries when feasible. Successful repair of TAA results from careful yet quick technique, as well as maintenance of optimal physiology by the anaesthesia and surgical teams. Distal aortic perfusion is accomplished either with left heart bypass and selective visceral perfusion or an axillary-femoral artery bypass before thoracotomy. Distal aortic perfusion manoeuvres are important for the prevention of major systemic morbidity following TAA repair.

Patients undergoing TAA repair frequently are older and have significant cardiac, pulmonary and other vascular comorbidities. These factors, combined with the magnitude of the operation and extent of aortic replacement, can lead to significant rates of mortality and serious morbidity. **[Q5]** Pulmonary complications remain most common and result from a combination of preoperative tobacco use, chronic obstructive pulmonary disease (COPD), and the effect of the thoracoabdominal incision on postoperative pulmonary mechanics. Reperfusion injury may also lead

to pulmonary microvascular injury and subsequent pulmonary dysfunction [8]. Cardiac complications remain the next most common, in spite of preoperative cardiac optimisation. Avoidance of hypotension, close monitoring perioperatively with pulmonary artery catheters, and minimisation of strain on the left ventricle can help decrease postoperative cardiac dysfunction. Using the bypass circuit to control ventricular afterload can reduce the risk of cardiac complications [9]. Renal insufficiency preoperatively increases the risk of postoperative renal failure. Minimising ischaemic time, selective renal perfusion during cross-clamping, distal aortic perfusion techniques, and avoidance of hypovolaemia are important in preventing renal failure [10].

Perhaps the most devastating complication following TAA repair is paraplegia. Despite years of research and development of protective strategies, paraplegia rates following TAA repair remain between 5 and 30 per cent, with an average of 13 per cent [6]. Risk factors for postoperative paraplegia include extent of aneurysm, cross-clamp time, postoperative hypotension, and oversewing of intercostal arteries. Cross-clamp times of less than 30 min are generally safe, while those in the range of 30–60 min are associated with increasing risk; cross-clamp times of more than 60 min carry the highest risk for neurological complications (Fig. 5.5). Minimising cross-clamp time and avoiding hypotension will decrease the risk of paraplegia. Sequential reperfusion of intercostal vessels by moving the cross clamp caudally as segments are reimplanted is useful to re-establish flow to these vessels quickly. In addition, avoiding prolonged mesenteric ischaemia, which may worsen reperfusion injury to the lungs, heart and possibly spinal cord through release of cytokines, is beneficial.

Numerous adjuncts have been studied for their ability to prevent paraplegia. **[Q6]** The use of cerebrospinal fluid (CSF) drainage to keep CSF pressure at less than 10 mm Hg has been shown to decrease the incidence of postoperative paraplegia when combined with distal aortic perfusion and/or moderate hypothermia [11].

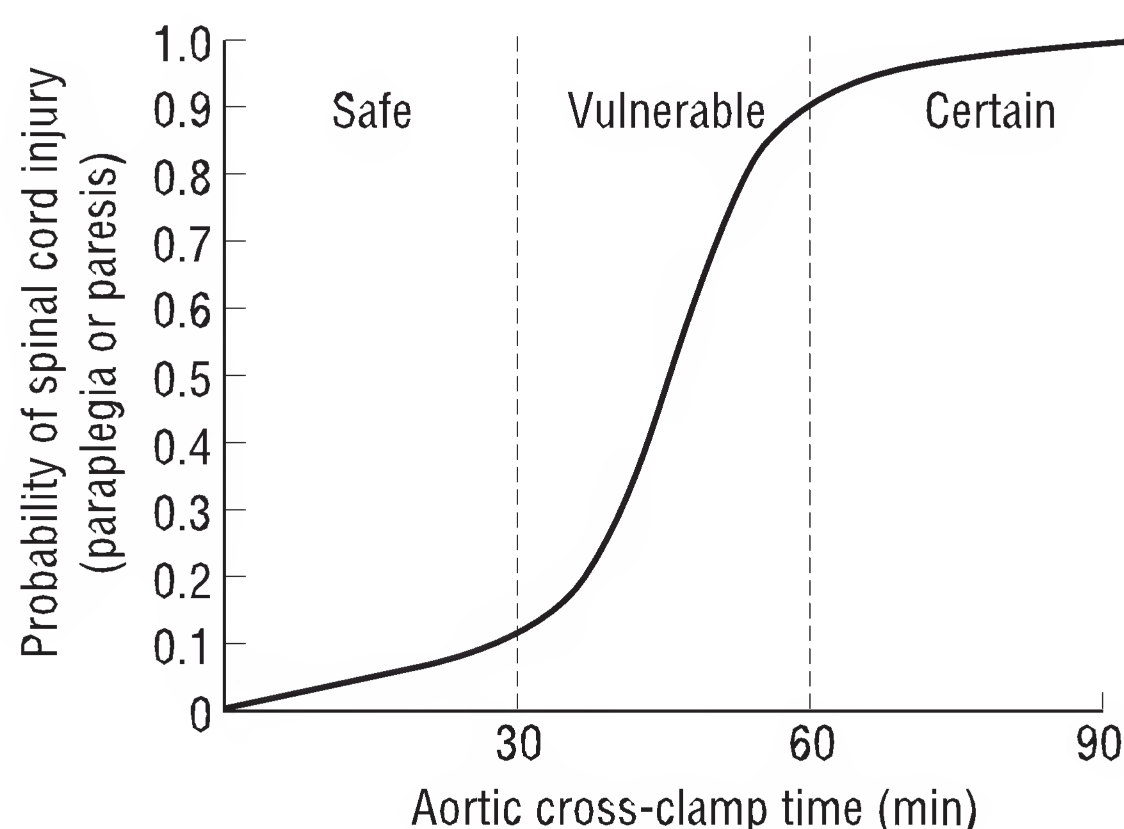


Fig. 5.5. Probability of postoperative paraplegia as a function of aortic cross-clamp time.

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Reimplantation of intercostal vessels is most likely beneficial in preventing postoperative paraplegia, provided this manoeuvre does not excessively prolong clamp time [12]. Epidural cooling by continuous infusion of cool saline via a catheter has been reported to decrease the incidence of paraplegia following TAA repair [13]. Preoperative angiographic localisation of the artery of Adamkiewicz followed by successful reimplantation of this vessel during surgery has resulted in no neurological sequelae in one series [14]. Patients who did not have preoperative localisation, or in whom reimplantation was unsuccessful, had a 50 per cent paraplegia rate. These results have not been reproduced, and angiographic localisation has not gained widespread acceptance. General anaesthetic agents can also help to prevent paraplegia, with propofol being the most protective. When left heart bypass is performed using pump techniques, moderate hypothermia can be used to protect the spinal cord. Other pharmacological adjuncts that may be beneficial include steroids and mannitol. Free-radical scavengers and inhibitors of excitatory neurotransmitter pathways have shown benefit experimentally but have not been proven clinically [15]. At present, the best strategy for preventing spinal cord complications appears to involve a combination of physiological optimisation of the patient perioperatively, intraoperative use of spinal drainage and some form of distal aortic perfusion, reimplantation of patent intercostal vessels, and minimisation of cross-clamp time. Other protective adjuncts are used based on surgeon preference and experience. Fig. 5.6 summarises the pathophysiology and prevention of neurological injury following TAA repair.

Some patients, as in the case we present here, will awake neurologically intact only to develop paraplegia hours to days later. **[Q8]** This phenomenon of delayed-onset paraplegia may represent reperfusion injury to areas of the spinal cord at risk from intraoperative hypoperfusion. Avoidance of postoperative hypoperfusion may decrease the incidence of this complication. The epidural catheter is left in place for 3 days postoperatively. In cases of delayed-onset paraplegia, maintenance of CSF pressure below 10 mm Hg may permit restoration of function. There are anecdotal reports of reversal of delayed-onset paraplegia by placement of an epidural catheter after onset of paralysis and removal of CSF to decrease pressure to below 10 mm Hg [16]. Lowering the CSF pressure may increase cord perfusion pressure enough to rescue the threatened regions of neuronal tissue. Lowering the CSF pressure to below 5 mm Hg may cause intracerebral haemorrhage, therefore the pressure must be monitored closely and maintained in the safe range.

Repair of a TAA represents a major physiological insult. Excellent anaesthesia care and monitoring are essential components of a successful operation. Postoperatively, large volumes of urine output must be replaced on a 1 : 1 basis in order to avoid hypovolaemia. Use of warmed, balanced electrolyte solutions is preferred. Hypocoagulability in the postoperative period is usually related to incomplete replacement of clotting factors and hypothermia. **[Q7]** In addition, supracoeliac aortic clamping has been shown to result in a state of fibrinolysis that may exacerbate bleeding [17].The aneurysm itself can be responsible for chronic coagulation factor consumption and a subsequent increased tendency to perioperative hypocoagulability [18]. Ongoing bleeding after TAA repair may require reoperation, and results in an increase in major morbidity and mortality. It is important to ensure that the PTT and partial thromboplastin times are corrected with plasma transfusions. Platelets should be replaced if thrombocytopoenia occurs in the face of ongoing bleeding. Hypothermia is a serious problem and can lead to failure of

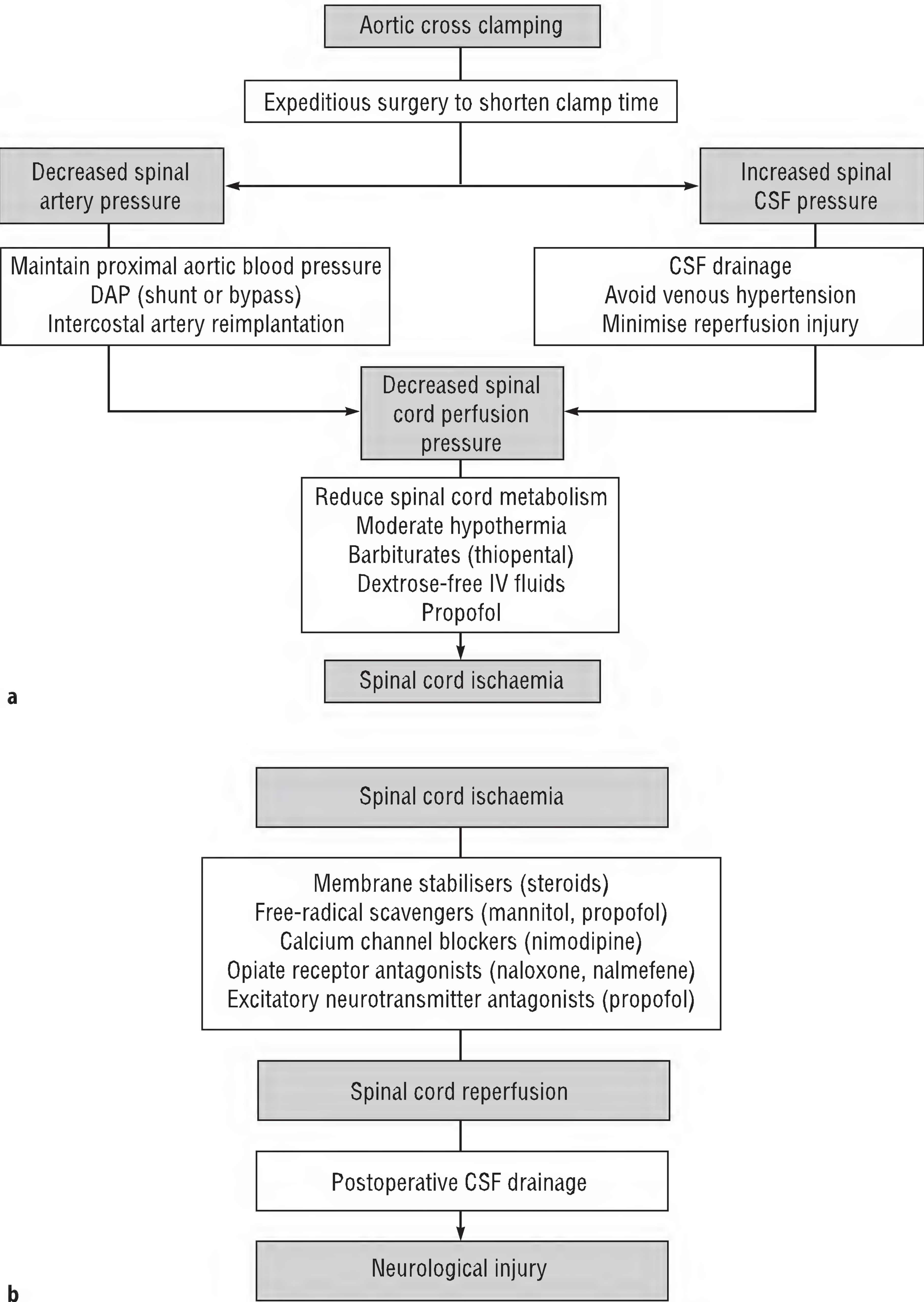


Fig. 5.6. Algorithm describing pathophysiology of spinal cord dysfunction following thoracoabdominal aneurysm surgery. Potential preventive interventions are also outlined.

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coagulation. Since hypothermia is often used intraoperatively as a spinal cord protective measure, it may persist as a problem postoperatively. Aggressive correction with warm fluids, blood products and warming blankets is needed to restore normothermia and proper function of coagulation as well as other enzymatic systems. Reoperation is reserved for ongoing significant bleeding following correction of coagulopathy and hypothermia. Reoperation for bleeding results in mortality rates of 25 per cent or greater in these patients [19].

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6. Aortic Dissection

Barbara T. Weiss-Müller and Wilhelm Sandmann

Dissection: Stanford A

A 68-year-old woman spontaneously and suddenly developed severe retrosternal pain during her holiday in Turkey. Without knowing the diagnosis, she flew home 2 days later. Computed tomography (CT) scans taken immediately after arrival revealed a dissection of the ascending aorta, the aortic bow and the descending aorta.

Question 1

How would you classify the aortic dissection?

A. Stanford A dissection.

B. Stanford B dissection.

C. de Bakey I dissection.

D. de Bakey II dissection.

E. de Bakey III dissection

On the same day, she underwent an emergency operation. The dissected ascending aorta with the entry of dissection was incised in a cardiopulmonary bypass and replaced by a graft using the in-graft technique. The aortic valve was patent and remained in situ. For reconstruction of the aortic root, the sandwich technique was used. Two Teflon strips were placed externally and into the true lumen to reattach the dissected membrane to the aortic wall. The aortic graft was then sutured into the reconstructed aortic root.

Question 2

Which of the following statements are wrong?

A. Stanford A dissections should be treated medically.

B. Stanford A dissections should undergo operation immediately.

C. Stanford B dissections without ischaemic complications should be treated medically.

D. Stanford B dissections require operative intervention immediately.

E. Stanford A dissections require an aortic stent graft immediately.

The postoperative course was uneventful at the beginning. However, 3 days later, renal function deteriorated and the patient required haemofiltration. Moreover, the patient developed severe hypertension and had to be treated with three different antihypertensive drugs. Contrast CT scans revealed that the right kidney was without function due to an old hydronephrosis, while the left renal artery was probably dissected. Furthermore, the patient developed left leg ischaemia and was transferred to our centre. We explored the abdomen via the transperitoneal approach. The pulsation of the left iliac artery was weak due to aortic and left iliac dissection. Infrarenal aorto-iliac membrane resection was performed to restore the blood flow to the extremities. Then the left renal artery was explored; the renal artery dissection was found to extend towards the hilus of the kidney.

Revascularisation was achieved with a saphenous vein interposition graft placed between the left iliac artery and the distal left renal artery (Fig. 6.1).

Question 3

Which of the following statements are correct?

A. Complications of Stanford A dissection are aortic valve insufficiency and perforation into the pericardium.

B. Stroke is a typical complication of Stanford B dissection.

C. Paraplegia is a typical complication of aortic dissection.

D. Most patients with Stanford B dissections die of aortic perforation.

E. Typical complications of aortic dissection are organ and lower-extremity ischaemia.

The postoperative course was uneventful. The patient recovered promptly from the operative intervention, while renal function and blood pressure improved substantially. Urine production and laboratory findings became normal, and only one antihypertensive drug (a beta-blocker) was necessary to maintain normal blood pressure. The postoperative angiography showed a patent iliac-renal interposition graft and normal perfusion of the left kidney (Fig. 6.1). CT scans taken 2 years later displayed a hypertrophic, well-functioning left kidney, while the right kidney was small and hydronephrotic (Fig. 6.2).



Fig. 6.1. a Left common iliac artery. **b** Left renal artery saphenous vein bypass.

Dissection: Stanford B

A 54-year-old woman was admitted to another hospital with the provisional diagnosis of a myocardial infarction (MI). She experienced a sudden chest pain. Some hours later, she developed paraesthesia in both legs, which improved spontaneously. Subsequently, she felt abdominal discomfort and developed diarrhoea and vomiting. The patient had been normotensive throughout her life, but now she required five different antihypertensive drugs to stabilise blood pressure. Some laboratory data were abnormal, including leucocytes, transaminases, lactic dehydrogenase and lactate. Duplex sonography and transoesophageal echocardiography revealed an aortic dissection of the thoracic and abdominal aorta beginning distal to the left subclavian artery; blood flow into the visceral arteries and the right renal artery was reduced. Contrast CT scans confirmed Stanford B aortic dissection.

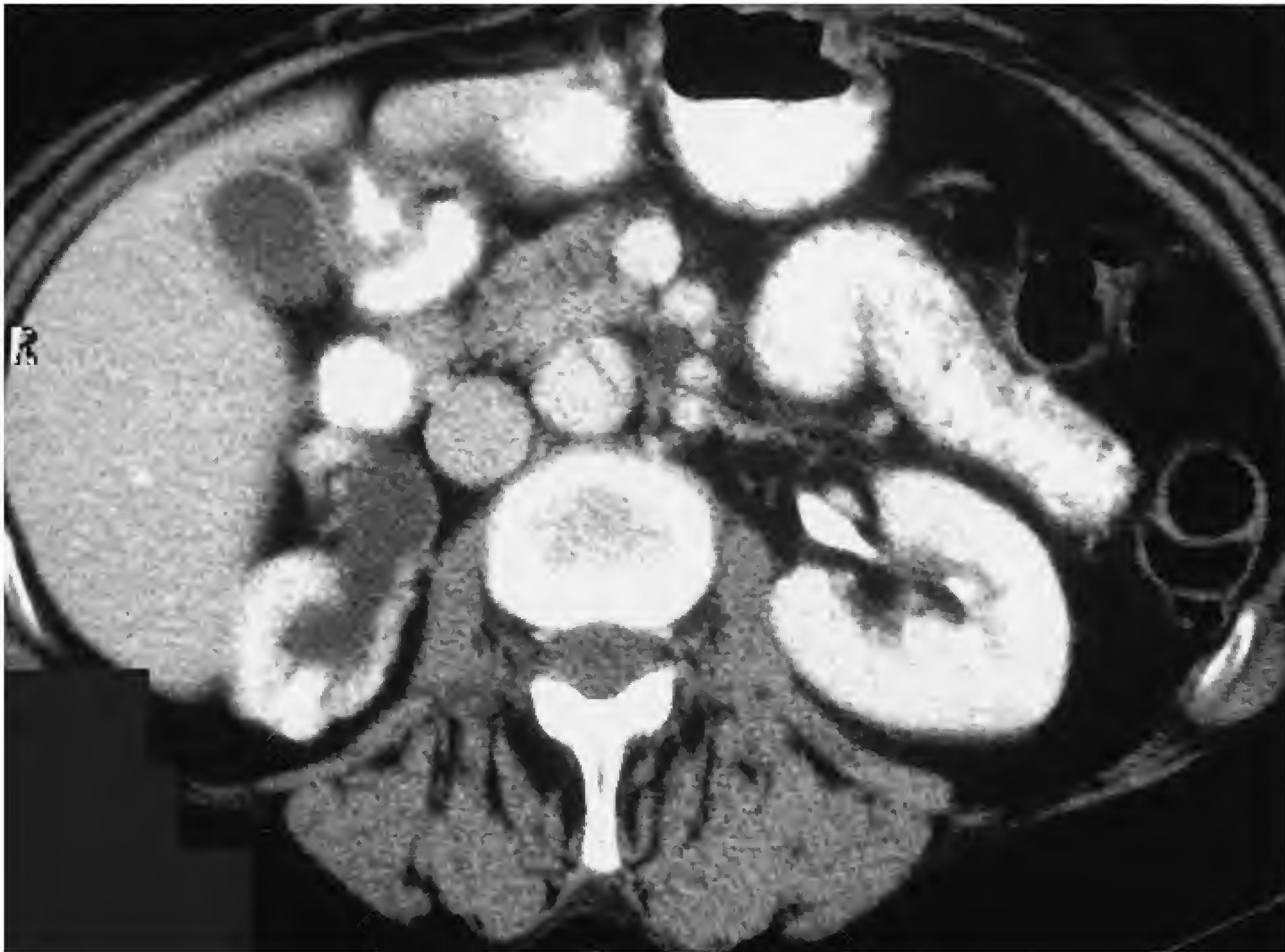


Fig. 6.2. CT scans taken 18 months after operative intervention show a well-functioning, hypertrophic left kidney and a small, hydronephrotic right kidney. Note the dissected but non-dilated aorta.

Question 4

What diagnostic methods are involved in acute aortic dissection?

A. Computed tomography.

B. Magnet resonance imaging.

C. Angiography.

D. Transoesophageal echocardiography.

The patient was first treated medically with parenteral therapy and antihypertensive drugs (including beta-blockers). Under this management, clinical outcome and laboratory findings improved, but 3 weeks later the patient deteriorated again and developed severe right upper abdominal pain.

She was referred to our hospital for operation. CT scans displayed the aortic dissection and a dissected superior mesenteric artery. The true aortic lumen was very small and partially thrombosed (Fig. 6.3). Abdominal exploration via the transperitoneal approach revealed borderline ischaemia of all intra-abdominal organs due to aortic dissection. The dissection had affected the coeliac trunk, the superior mesenteric artery and the right renal artery. The right upper abdominal pain was caused by an ischaemic cholecystitis. The gallbladder had to be removed. The para-aortic tissue displayed severe inflammation; therefore no fenestration and membrane

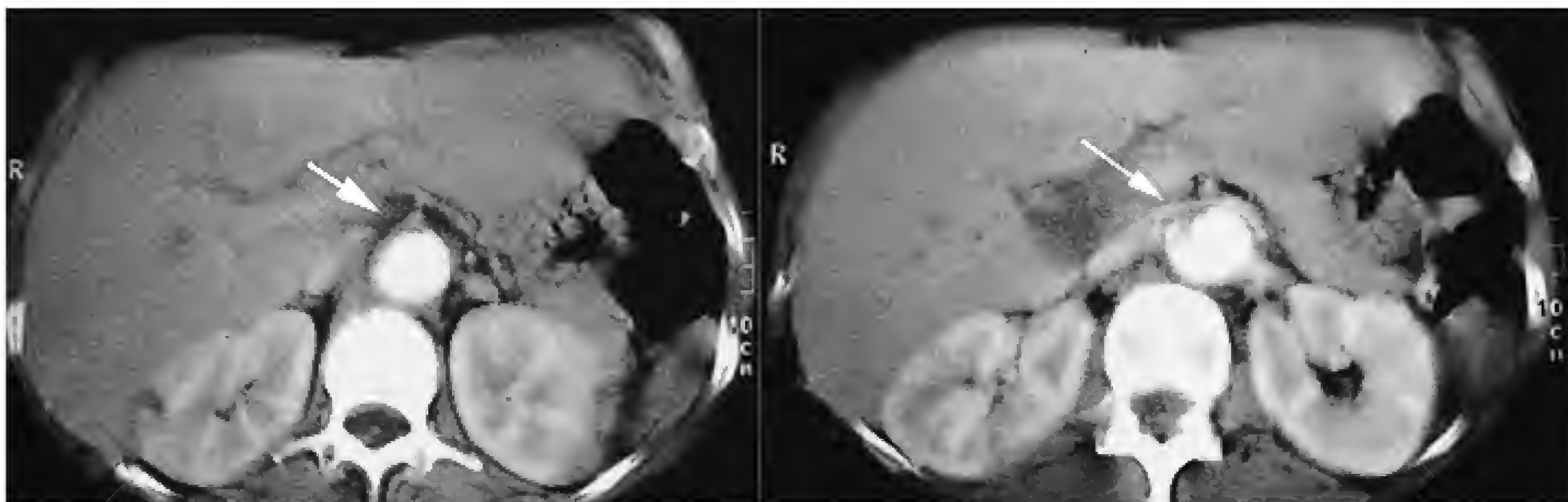


Fig. 6.3. Aortic dissection, with a small, partially thrombosed "true" aortic lumen and dissected superior mesenteric artery. 📖

resection could be carried out. Instead, intestinal and renal blood flow was restored by a 12-mm Dacron graft, which was placed end to side into the left iliac artery and end to end to the coeliac trunk. The superior mesenteric artery was implanted directly into the Dacron graft, while the right renal artery was attached by means of a saphenous vein interposition graft (Fig. 6.4).

Question 5

What techniques are used to restore blood flow to the visceral organs and extremities following ischaemia from aortic dissection? Which of the following statements are wrong?

A. Aortic stent graft.

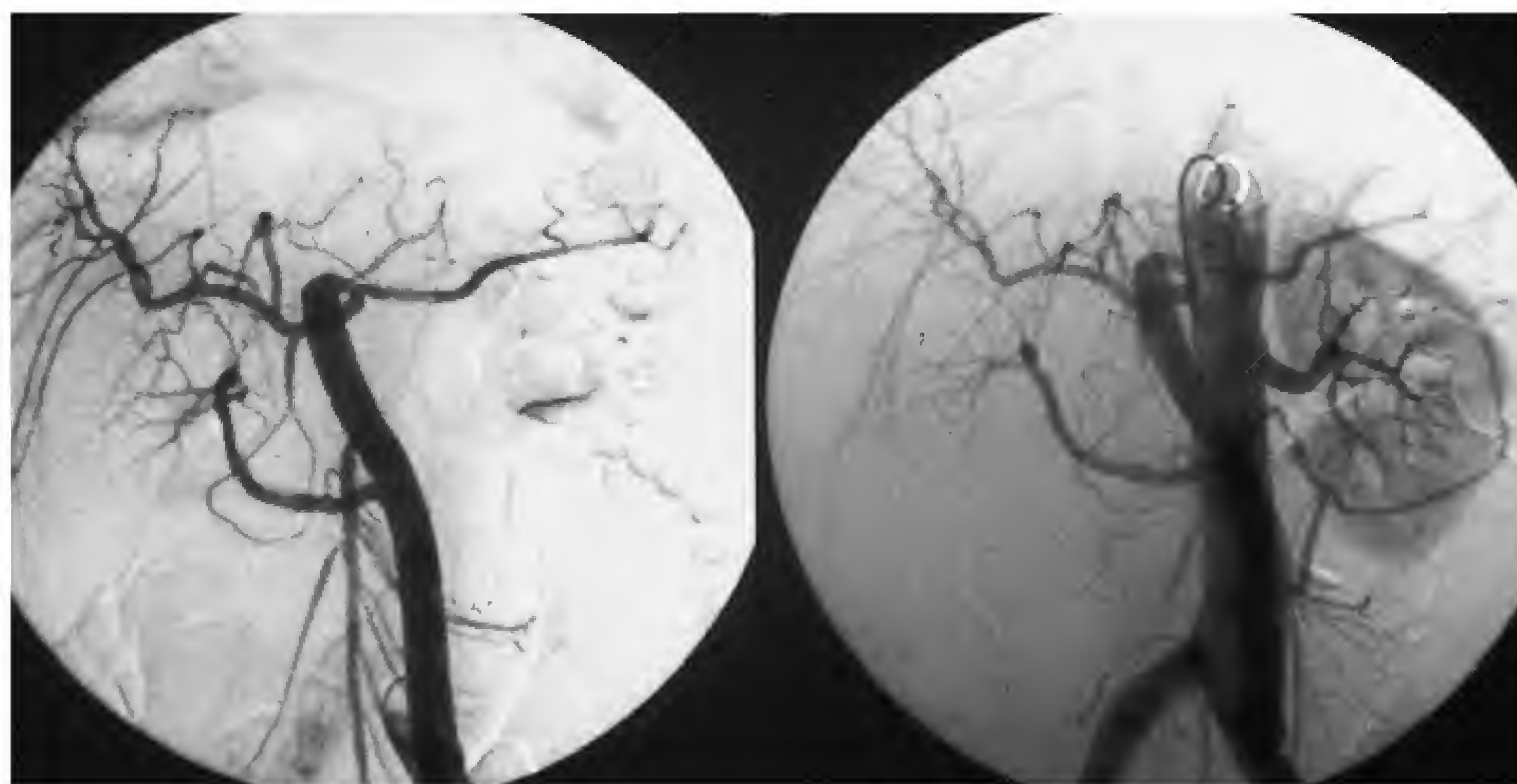


Fig. 6.4. Extra-anatomical reconstruction with a Dacron graft, which was placed end to side between the left common iliac artery and end to end to the coeliac trunk. The superior mesenteric artery was implanted directly into the graft, while the right renal artery was implanted via the interposition of a saphenous vein. The left renal artery originates from the aorta. 📖

B. PTA of organ and limb arteries and stenting.

C. Aortic fenestration and membrane resection.

D. Cardiopulmonary bypass.

E. Extra-anatomic revascularisation, e.g. axillo-femoral bypass.

The complication of postoperative retroperitoneal bleeding from the reconstructed right renal artery had to be managed by relaparotomy and single vascular stitches, and clinical recovery was delayed. The patient required 4 months of rehabilitation until she had regained her previous health status. At this point, digestion and renal function had recovered, laboratory findings became normal, and hypertension had to be treated with only one drug (beta-blocker). Postoperative angiographies showed good perfusion of all visceral and renal arteries via the Dacron graft (Fig. 6.4).

Commentary

The life-threatening aortic dissection starts with an intimal tear (entry) in the ascending aorta (Stanford A, de Bakey I or II) or distally to the left subclavian artery (Stanford B, de Bakey III). De Bakey II dissection affects the ascending aorta only, while de Bakey I and III dissections also involve the descending aorta [1, 2]. **[Q1: A, C]** Most patients with acute aortic dissection present with severe chest pain, which might be misinterpreted as acute MI [3, 4].

Echocardiography, particularly by the transoesophageal approach, is a reliable and rapid method for diagnosis of aortic dissection and differentiation into Stanford A or B type [5]. Nevertheless, the evaluation of organ arteries and their blood flow by ultrasound may be difficult in acute dissection. In our opinion, contrast thoracic and abdominal CT scans, especially using the spiral technique, are appropriate diagnostic methods for determining the extension of dissection and the relation of its dissecting membrane to major branches of the aorta. The perfusion of abdominal organs, and often of their arteries, can be seen easily. In the case of organ malperfusion, angiography may be helpful to determine whether the ischaemia is caused by the dissecting membrane of the aorta or whether the dissection extends into the organ arteries [6]. Magnetic resonance imaging (MRI) or magnetic resonance angiography (MRA) are effective alternatives in the diagnosis of patients with dissection and renal failure [7]. **[Q4: A–D]**

Without treatment, the prognosis of acute aortic dissection is very poor. In 1958, Hirst et al. reviewed 505 cases of aortic dissection and found that 21 per cent of patients died within 24 h of onset and only 20 per cent survived the first month [3]. Causes of death in patients with Stanford A dissection include intrapericardial and free intrapleural rupture, acute aortic valve insufficiency, and, to a minor extent, cerebral and coronary malperfusion. In patients with type B dissections, free rupture of the aorta is less frequent. Dissection of the descending aorta may lead, in about 30 per cent of cases, to obstruction of visceral, renal and extremity arteries, resulting in visceral ischaemia, renal insufficiency and acute limb ischaemia, which may be lethal without prompt and adequate therapy [8–10]. **[Q3: A, C, E]**

To improve the natural course of the disease, in 1955 de Bakey et al. started to treat acute aortic dissections surgically. Within only a few years, they had developed

the current principles of operative intervention in acute Stanford A dissection with replacement of the ascending aorta by a graft in cardiopulmonary arrest. Their results were outstanding, with an overall mortality of 21 per cent [1, 11].

However, the surgical experiences of other workgroups were not so successful. Therefore, Wheat et al. developed a new medical treatment with ganglionic blockers, sodium nitroprusside or beta-blockers to influence the hydrodynamic forces of the bloodstream based on the theory that blood pressure and the steepness of the pulse wave are propagating the dissecting haematoma [12]. In 1979, a meta-analysis of 219 patients with acute aortic dissection from six centres revealed that Stanford A patients treated medically had a mortality of 74 per cent, whereas 70 per cent of patients survived after surgical therapy. On the other hand, in patients with acute type B dissection, drug therapy alone had a survival rate of 80 per cent, whereas 50 per cent died after operative intervention [13]. Therefore in most centres, current therapy for acute dissection type Stanford A is surgical [14–17], and for uncomplicated Stanford B dissection it is medical [18–22]. **[Q2: A, D, E]**

An acute dissection, involving the ascending aorta, should be considered a surgical emergency. The aim of operative intervention is to prevent or treat dilation or rupture of the aortic root, and to maintain aortic valve function. The following reconstructive approach is recommended: in patients in whom the root is not involved by dissection, a tubular graft is anastomosed to the sinotubular ridge. In the presence of commissural detachment, the valve is resuspended before supracommissural graft insertion. If the aortic valve is affected by congenital or acquired abnormalities, then it is generally replaced [15].

Patients with acute uncomplicated Stanford B dissection should be treated medically. Careful monitoring is obligatory, while antihypertensive drugs, such as beta-blockers [23], and analgesics are administered. The aim of treatment is to stabilise the dissected aortic wall within 2 weeks and to prevent further extension of dissection or perforation. Careful clinical and laboratory examinations are necessary to detect symptoms of organ or extremity malperfusion in time. Limb, renal and visceral ischaemia can be observed frequently, but paraplegia due to malperfusion of intercostal arteries is rare [6, 8–10].

If peripheral vascular complications occur, several therapeutic strategies are possible. Newer publications describe endovascular procedures, for example emergency aortic stenting to close the "entry" and the false aortic lumen [24–27]. Ultrasound-guided endovascular catheter aortic membrane fenestration was performed to restore the blood flow to the aortic branches. Dilation and stenting of dissected organ or iliac arteries were performed to resolve stenosis and restore blood flow [28–30]. These new therapeutic methods need to be evaluated in long-term follow-up.

Aortic surgery in the acute stage of aortic dissection is a dangerous procedure. The dissected aortic wall is extremely friable and does not hold sutures well. Therefore we, and many other centres, try to leave the aorta itself untouched and to restore organ or extremity blood flow by extra-anatomical bypass procedures. Extra-anatomical revascularisation also becomes necessary if the aortic branches themselves are dissected [6, 8, 15]. Normally, we use one common iliac artery as the donor vessel for extra-anatomical bypass grafting, but the distal lumbar aorta might also be suitable. If only one aortic branch requires revascularisation, then the iliac-visceral bypass is performed with the saphenous vein (Fig. 6.1). If two or more branches are affected, then a Dacron graft is used and the visceral arteries can be implanted into the graft directly or via interposition of the saphenous vein

(Fig. 6.4). Blood flow to the legs can be restored with a femoral-femoral crossover bypass or with an axillo-(bi)-femoral graft. If several organ arteries are occluded by the aortic dissecting membrane, and the visceral arteries are undissected, then abdominal aortic fenestration and membrane resection combined with thrombectomy of the organ arteries can also be performed [31–34]. We prefer the latter to treat paraplegia caused by acute aortic dissection. **[Q5: D]**

Our only indication for total aortic replacement in the acute stage of dissection is aortic penetration or perforation.

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7. Popliteal Artery Aneurysm

Jonathan D. Woody and Michel S. Makaroun

A 77-year-old male presented to the emergency department with a 2-day history of increased swelling and pain in the left knee and distal thigh. He was unable to extend the knee without severe pain. On examination, the patient had a large pulsatile mass in the left popliteal fossa and distal thigh.

Question 1

Which of the following is the initial diagnostic test of choice for popliteal artery aneurysm?

A. Magnetic resonance imaging (MRI).

B. Arteriography.

C. Duplex ultrasonography.

An arteriogram was obtained, which revealed bilateral popliteal artery aneurysms, right common iliac artery aneurysm, and multiple aneurysms of the left internal iliac artery (Figs 7.1 and 7.2).

Question 2

Which of the following statements about popliteal artery aneurysm is false?

A. It is bilateral in approximately 60 percent of cases.

B. Approximately 80 percent of patients with bilateral popliteal artery aneurysm have another aneurysm elsewhere.

C. Duplex ultrasonography is the initial diagnostic test of choice.

D. Rupture of popliteal artery aneurysm occurs frequently.

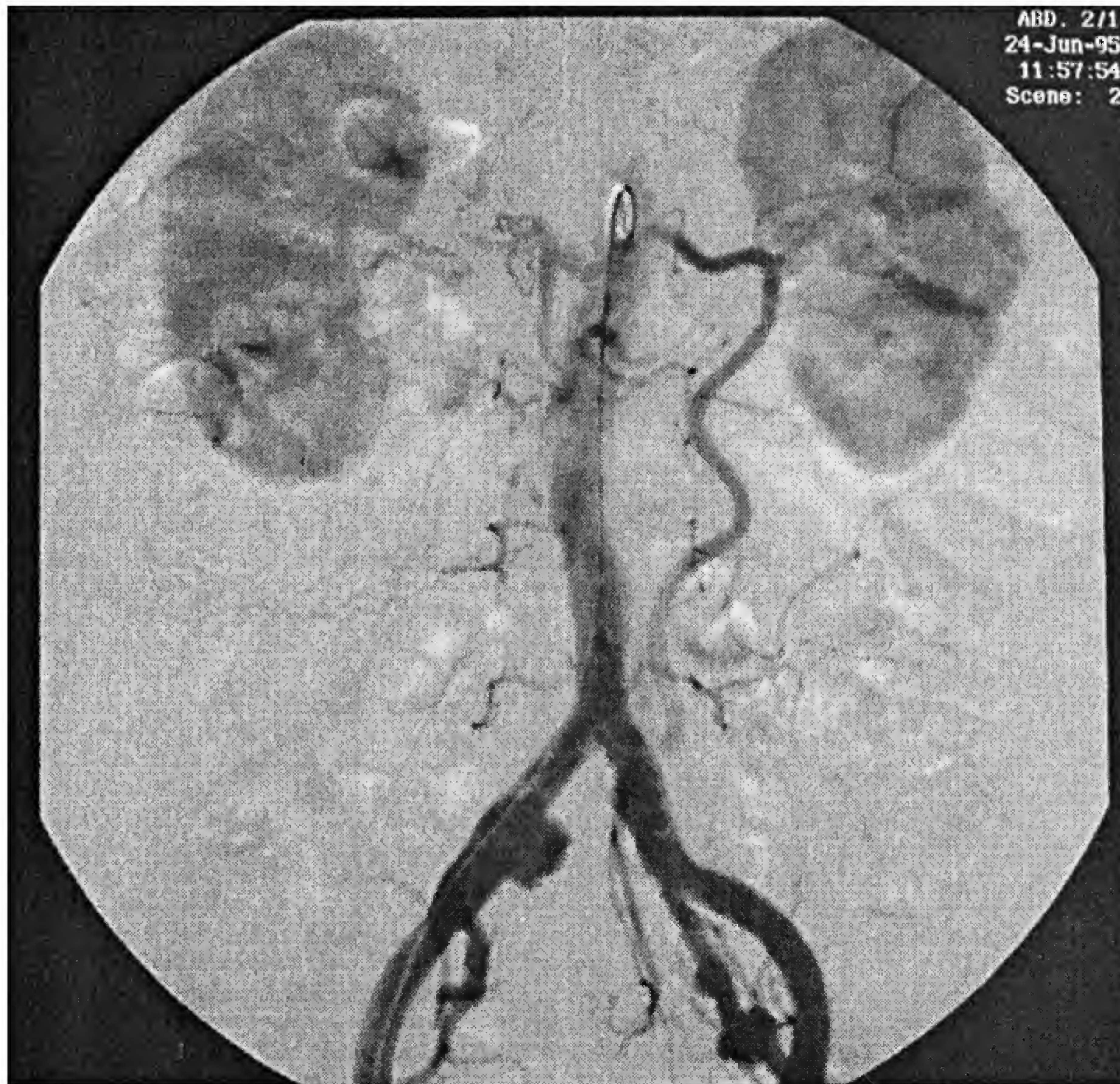


Fig. 7.1. Arteriogram demonstrating aneurysms of the right common iliac artery and left internal iliac artery. 📖

Question 3

Which of the following are indications for operative treatment of popliteal artery aneurysm?

A. Size greater than 2 cm.

B. Swelling or pain in the affected leg.

C. Aneurysm of any size with mural thrombus.

D. Distal embolization.



Fig. 7.2. Arteriogram demonstrating bilateral popliteal artery aneurysms.

Treatment

The patient underwent operative exploration of the left popliteal fossa through a medial approach. After proximal and distal control was obtained, the aneurysm was opened and all collateral flow into the aneurysm was obliterated. An interposition vein graft was placed from the distal superficial femoral artery to the distal popliteal artery. The patient subsequently underwent surgical treatment of the right popliteal artery aneurysm and endovascular treatment of the right common iliac artery aneurysm.

Question 4

Which of the following statements regarding the treatment of popliteal artery aneurysm is false?

A. Bypass and exclusion of the aneurysm is safe and effective.

B. Resection and endoaneurysmorrhaphy is safe and effective.

C. The medial approach is the preferred route of exposure.

D. Endoluminal stent grafting may be indicated in a high-risk patient.

E. For acute thrombosis, surgical thrombectomy has a higher limb salvage rate than thrombolysis followed by surgical reconstruction.

Question 5

All of the following are true regarding the treatment of acute thrombosis of a popliteal artery aneurysm except:

A. Limb loss occurs in up to 50 percent of cases.

B. Thrombolysis improves the run-off and increases limb salvage.

C. Thrombolysis alone is adequate treatment.

D. Surgical thrombectomy is inferior to thrombolysis followed by surgical reconstruction.

Commentary

Popliteal artery aneurysm is the most common type of peripheral aneurysm; the vast majority are caused by atherosclerosis. The typical patient is a male in his sixth or seventh decade. Popliteal artery aneurysm occurs bilaterally in approximately 60 percent of cases [1]. Approximately 80 percent of patients with bilateral popliteal artery aneurysm have other aneurysms elsewhere, and 1 percent of patients with abdominal aortic aneurysm (AAA) have popliteal artery aneurysm [2].

Diagnosis of popliteal artery aneurysm is relatively straightforward. Examination reveals a prominent pulsation or a pulsatile mass in the popliteal fossa. Duplex ultrasonography is the initial diagnostic test of choice because it can differentiate an aneurysm from other masses in the popliteal fossa and it can determine whether thrombus is present in the aneurysm. **[Q1: C]** Angiography is not sensitive for the documentation of an aneurysm, but it is crucial for evaluating the status of the distal arterial system. If the diagnosis can be made on physical examination, then one can proceed directly to angiography for operative planning.

The majority of patients with popliteal artery aneurysm are symptomatic at the time of diagnosis. Distal embolization is the most common form of presentation, followed by pain and/or swelling of the affected extremity due to compression of the

adjacent nerves and veins. Rupture is rare and usually not life threatening. **[Q2: D]** However, rupture can lead to limb loss in 50–75% of cases due to associated limb ischemia [3].

Indications for operation include size of 2 cm or greater, aneurysm of any size with mural thrombus, any patient with pain and/or swelling due to compression of adjacent structures, and any symptoms of embolization. While some surgeons prefer to delay operation in asymptomatic patients until the aneurysm reaches 3 cm in size, we believe that an aggressive approach is justified because the majority of patients ultimately become symptomatic. Some authors advocate an aggressive approach to any small, asymptomatic popliteal artery aneurysm containing mural thrombus due to the high frequency of embolization in these cases [4]. **[Q3: A, B, C, D]** It is important to note that thromboembolization is the most common form of presentation. Accepted forms of surgical treatment include bypass with complete exclusion of the aneurysm, bypass with partial or complete resection of the aneurysm if compressive symptoms are present, or endoaneurysmorrhaphy like the open repair of AAA. The medial approach is preferred over the posterior approach because it allows for better exposure of the distal superficial femoral artery and tibioperoneal trunk, easier access to the saphenous vein, and the ability to perform distal bypass if it becomes necessary. Autogenous vein is the preferred conduit since it provides better patency than prosthetic graft. Endoluminal stent grafting has not been proven, and theoretically it would seem to be inferior to surgical repair due to the small size of the stent graft required and the lower patency rates of prosthetic graft in this position. Nevertheless, it may be an option in a patient who is at high risk for a surgical procedure.

Distal embolization can cause tissue loss, but the more troubling consequence is the destruction of the run-off bed. This results in decreased patency of bypass grafts and an increased rate of limb loss. Further, there is a significant reduction in limb salvage and graft patency in symptomatic patients compared with asymptomatic patients. Numerous studies indicate that virtually all patients will become symptomatic and that graft patency and limb salvage approach 100 percent in elective reconstruction [1–6]. This demonstrates the increased risk of complications and limb loss in untreated patients and underscores the importance of early treatment.

Acute thrombosis is a particularly vexing problem. While some may present with only claudication due to the previous development of collateral flow, many present with threatened limb loss. In the setting of acute thrombosis, the combined rate of primary and early amputation is 50 percent and operative mortality is 5 percent [7]. Thrombolysis followed by surgical treatment yields better graft patency and limb salvage than surgical thrombectomy. **[Q4: E]** Most likely, this is due to some degree of clearing of the run-off bed. Thrombolysis alone is inadequate treatment since surgical reconstruction is required to obliterate the aneurysm. **[Q5: C]**

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8. Renal Artery Aneurysm

Lutz Reiher, Tomas Pfeiffer and Wilhelm Sandmann

A 45-year-old woman presented with a 10-year history of arterial hypertension. After initially successful conservative therapy with two antihypertensive drugs, arterial blood pressure was not controlled well during the last months. To exclude a renovascular origin of hypertension, an angiography was performed, which showed fibrodysplastic disease of the right renal artery with several stenotic segments and aneurysms (Fig. 8.1).

Question 1

Which of the following statements regarding renal artery aneurysm (RAA) is correct?

A. It has a marked female preponderance.

B. It is usually diagnosed during examination for flank pain.

C. It may cause arterial hypertension.

D. It typically leads to proteinuria by compression of the renal vein.

E. It can cause haematuria in rare cases.

Question 2

Which statements about the aetiology of the RAA are true ?

A. The most frequent underlying diseases of RAA are aortic coarctation with concomitant disease of the renal artery and renal artery dissection.

B. Fibromuscular dysplasia of the renal artery may present with renal artery stenosis (RAS), RAA or both.

C. Arteriosclerosis is a frequent cause of RAA.

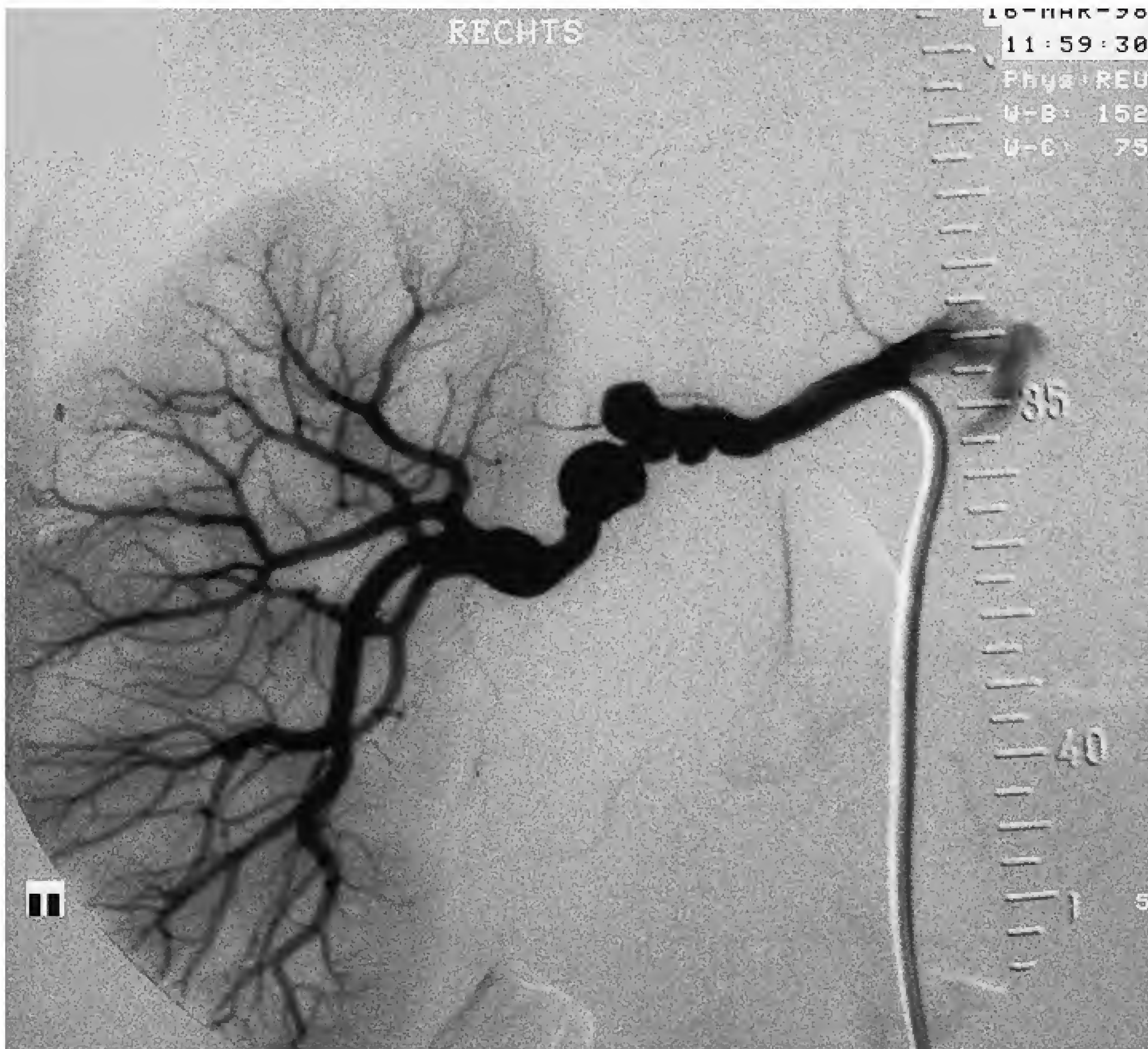


Fig. 8.1. Selective intra-arterial renal artery angiography revealed RAA combined with renal artery stenosis due to fibromuscular dysplasia.

D. Some RAA present with inflammation of the arterial wall.

E. The incidence of RAAs is increased in Ehlers–Danlos syndrome and Marfan's syndrome

Question 3

Which risks of the spontaneous course of the RAA should you explain to your patient?

A. The RAA may rupture and lead to a life-threatening bleeding.

B. The risk of rupture decreases during pregnancy and childbirth.

C. Hypertension in RAA may be caused by concomitant stenosis of the renal artery or its branches.

D. In cases of RAA and hypertension the angiography of the renal artery always shows an additional RAS.

E. The RAA may be a source of embolisation leading to a loss of renal function.

Question 4

Which of the following statements regarding the indication of renal artery repair (RAR) for RAA is correct?

A. There is an indication for RAR only in cases of symptoms other than hypertension.

B. There is no reason to perform RAR in women of childbearing age if there is no arterial hypertension.

C. There is a good indication for RAR if a concomitant RAS is found.

D. There is a good indication for RAR only if the RAA is larger than 5.5 cm.

E. There is an indication for RAR in patients presenting with RAA and hypertension even if an additional RAS is not detectable.

For RAR, a midline abdominal incision was performed for direct access to the infrarenal aorta, where an end-to-side anastomosis was performed with a segment of the patient's greater saphenous vein. After Kocher's manoeuvre, the distal renal artery was transected and anastomosed to the saphenous vein, which had been placed on the renal hilus dorsal to the inferior vena cava. Good results were shown by postoperative angiography (Fig. 8.2). At re-examination 3 years after the operation, the patient had a normal blood pressure without antihypertensive medication.

Question 5

Which of the following statements regarding the management of RAA is correct?

A. Replacement of the diseased renal artery by prosthetic graft is the RAR of first choice.

B. Protection of the kidney against ischaemic injury is performed only during ex situ reconstruction of the renal artery.

C. RAA exclusion and aortorenal vein graft interposition, or RAA resection and end-to-end anastomosis or aneurysmorrhaphy, are valuable methods for RAR.

D. Ex situ repair of the renal artery may be needed in cases presenting with lesions of the distal branch arteries.

E. Tailoring of RAA often leads to recurrent aneurysmatic dilation of the renal artery.

Commentary

RAAs do not usually cause symptoms, and generally they are diagnosed accidentally during work-up for hypertension, as in our patient. In rare cases, flank pain has

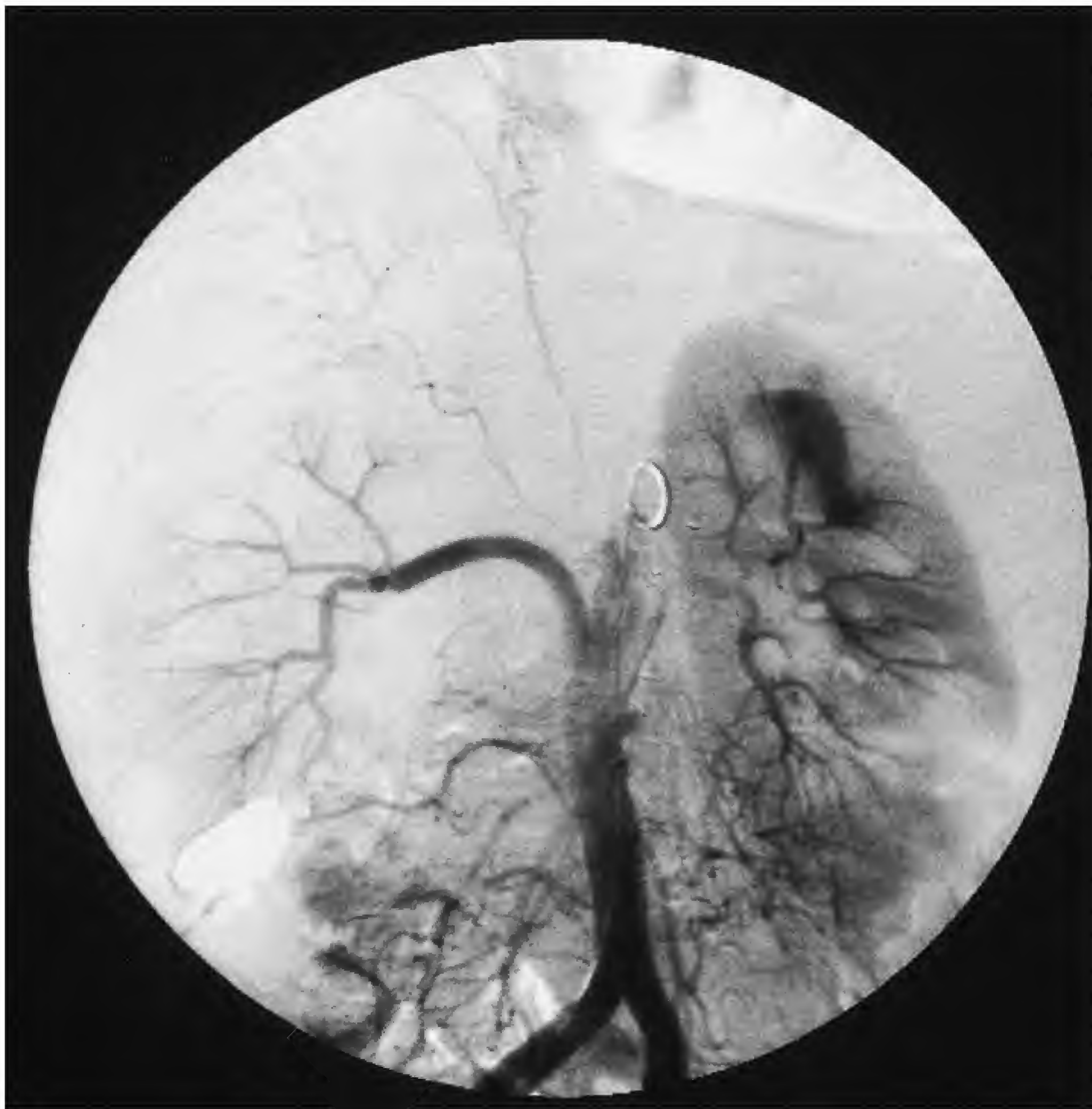


Fig. 8.2. Postoperative angiography demonstrates a patent aortorenal venous graft. 📖

been described as the initial symptom, which may be due either to the size of the RAA or to a renal artery dissection. Rupture of the aneurysm into the urinary tract will lead to haematuria. **[Q1: A, C, E]** The underlying disease is most frequently dysplasia of the arterial wall followed by arteriosclerosis. In our case, fibromuscular dysplasia was found to be the aetiology of the RAA. Rare causes of RAA may be atypical aortic coarctation with concomitant disease of the renal arteries, inflammation of the arterial wall, dissection or trauma, or disorders of the elastic and collagen fibres (i.e. Ehlers–Danlos syndrome or Marfan's syndrome). **[Q2: B, C, D, E]**

RAA is found about twice as often in the right renal artery as in the left. Selective angiography often reveals concomitant RAS of mainstem and segmental arteries, and segmental arteries may also be aneurysmal. Concomitant renal artery dissection is rare.

Rupture of RAA, development or deterioration of arterial hypertension, and loss of renal function by thrombosis or embolisation, are impending spontaneous consequences of RAA.

As with all arterial aneurysms, rupture is a possible complication of RAA. While Tham et al. [1] experienced no rupture of RAA in 69 patients who had been treated conservatively during a mean observation time of 4.3 years, Henriksson et al. [2] observed RAA rupture in four cases (10.2 percent), and at the time of rupture only a nephrectomy could be performed. There are several case reports about RAA rupture in pregnancy and childbirth [3–5], and one author found the probability of RAA rupture during pregnancy to be as high as 80 percent [6].

As high arterial blood pressure is in itself a risk factor for rupture of arterial aneurysms of any localisation, one can argue that hypertension per se is an indication to remove an RAA. Hypertension was found in 90 percent of all patients with ruptured RAA [7].

The larger the diameter of the RAA, the more likely the danger of rupture seems to be, which can be explained by Laplace's law. However, RAAs of any diameter can rupture. In one patient cohort [8], the smallest (1 cm) and the largest (16.5 cm) RAAs ruptured.

About eighty percent of patients with RAA have arterial hypertension [9, 10]. If RAA is accompanied by RAS on the same or the contralateral side, as in our patient, then it is reasonable to remove both, with the intention to improve hypertension and eliminate the risk of rupture. However, an ipsilateral stenosis may be missed by angiography due to overprojection of the aneurysm. Furthermore, aneurysmal disease includes not only dilation of vessels but also elongation, which might cause kinking with a relevant stenosis [11]. **[Q3: A, C, E]**

There is an absolute indication to remove RAAs in all patients with arterial hypertension with and without concomitant RAS and in women of childbearing age. **[Q2: C]** RAAs with a diameter greater than 2 cm should be removed, even if there is no hypertension. There are good long-term results for autologous RAR; therefore, there is a relative indication for operation in younger patients without hypertension and concomitant RAS with RAA of diameter of 1 cm or more. **[Q4: C, E]**

The most promising method of RAR is by autogenous reconstruction. Methods of RAR are replacement of the renal artery by the greater saphenous vein, resection of diseased sections and reanastomosis. The autoplastic reconstruction by tailoring (synonym: aneurysmorrhaphy) is another appropriate technique. Although the aneurysmatic wall is only resected partially, recurrent RAAs have not been observed. The in situ reconstruction is less traumatic, but ex situ repair of the renal artery may be necessary in cases in which not only the distal mainstem artery but also the segmental arteries are involved. **[Q5: C, D]**

If arterial repair is restricted to renal arteries only, and if concomitant repair of the aorta is not necessary, then a postoperative mortality of less than 1 percent can be expected. Postoperative morbidity is due to temporary kidney insufficiency, graft thrombosis, bleeding, thrombosis and pancreatitis. Affected kidneys can be preserved in more than 85 percent of cases. The number of patients who benefit from surgical therapy in terms of improvement of arterial hypertension varies considerably between authors, ranging from 5 to 50 percent and from 25 to 62 percent, respectively [12].

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9a. Anastomotic Aneurysms

William D. Neary and Jonothan J. Earnshaw

A 70-year-old woman presented with bilateral pulsatile groin masses (Fig. 9a.1). Six years ago, she had an elective aorto-bifemoral graft for a 6-cm abdominal aortic aneurysm involving both iliac arteries, from which she made a full recovery. She first found the larger, right-sided mass 4 months ago, and she had noted gradual enlargement since then. She had no symptoms of claudication or leg ischaemia. Her past medical history included a myocardial infarction (MI) 18 months ago, but without limitation to her exercise tolerance.

On examination, the patient appeared well. There was a well-healed midline laparotomy scar from the previous operation. Abdominal examination was unremarkable, and there were no bruits on auscultation. Two well-defined expansile masses were palpable in the middle third of the femoral scars, measuring approximately 2 cm on the left and 4 cm on the right. The masses were not tender. There was no evidence of compromise in the distal circulation, and all pulses were palpable. Duplex imaging was performed, which identified anastomotic false aneurysms in both groins, measuring 1.8 cm on the left and 3.5 cm on the right.

Question 1

Which of the following statements regarding the aetiology of anastomotic false aneurysms are correct?

A. Anastomotic false aneurysms occur in 3–5 per cent of anastomoses to the femoral artery in the groin.

B. Seventy per cent are found in the groin.

C. Primary degeneration of the arterial wall is an aetiological factor.

D. Continued smoking is an aetiological factor.

E. At reoperation, approximately one-third will be found to be infected with pathogenic bacteria.



Fig. 9a.1. Female patient with bilateral anastomotic aneurysms from an aortobifemoral graft.

Question 2

The patient wished to know the risks of leaving the aneurysm alone. Rank the potential complications of anastomotic aneurysms in order of frequency.

A. Rupture.

B. Embolisation.

C. Pressure symptoms.

D. Pain.

E. Secondary haemorrhage.

Question 3

Which of the following non-operative treatments are also available?

A. Embolisation.

B. Ultrasound-guided compression.

C. Thrombin injection.

D. Intravascular stent graft.

The larger of the two aneurysms was repaired surgically. The previous surgical incision was reopened and extended. A large false aneurysm was confirmed; the graft appeared to have become detached from the artery. There were no signs of infection. The aneurysm was replaced by straight 8-mm gelatine-coated woven

Dacron interposition graft (soaked in rifampicin solution 10 mg/ml) taken end to end from the old graft and sutured end to side over the common femoral bifurcation. The thrombus and old graft were sent for microbiology. The patient made a good postoperative recovery. All bacterial cultures were negative, so perioperative antibiotic prophylaxis was stopped after 48 h.

Question 4

Rank the following surgical procedures in order of use for the management of anastomotic aneurysm in the groin:

A. Resuture or local repair.

B. Ligation and bypass.

C. Prosthetic patch.

D. Vein patch.

E. Interposition graft.

This patient at 2-year follow-up had no evidence of recurrence of the pseudoaneurysm in her right groin. An ultrasound scanning of her left groin revealed that the left pseudoaneurysm remained 2 cm in maximum diameter.

Question 6

The following statements are true or false.

A. Surgery cures 50 per cent of all anastomotic aneurysms.

B. Surgery cures 90 per cent of all anastomotic aneurysms.

C. Surgery cures 50 per cent of all recurrent anastomotic aneurysms.

D. Surgery cures 90 per cent of all recurrent anastomotic aneurysms.

E. Long-term follow-up of retroperitoneal anastomotic aneurysms is not necessary.

Commentary

The incidence of anastomotic aneurysms is increasing, due primarily to the increased frequency of prosthetic vascular reconstructions involving groin anastomosis. The overall incidence following vascular anastomoses is about 2 per cent, but this increases to 3–8 per cent when the anastomosis involves the femoral artery [1–4]. Although they are most common after prosthetic bypass, anastomotic aneurysms occasionally occur after vein bypass, semi-closed endarterectomy, and open endarterectomy with a vein patch. Anastomotic aneurysms can occur anywhere, but they frequently develop near to a joint. About 80 per cent occur at the groin [1], presumably due to movement-related strains. **[Q1: A, C, D, E]**

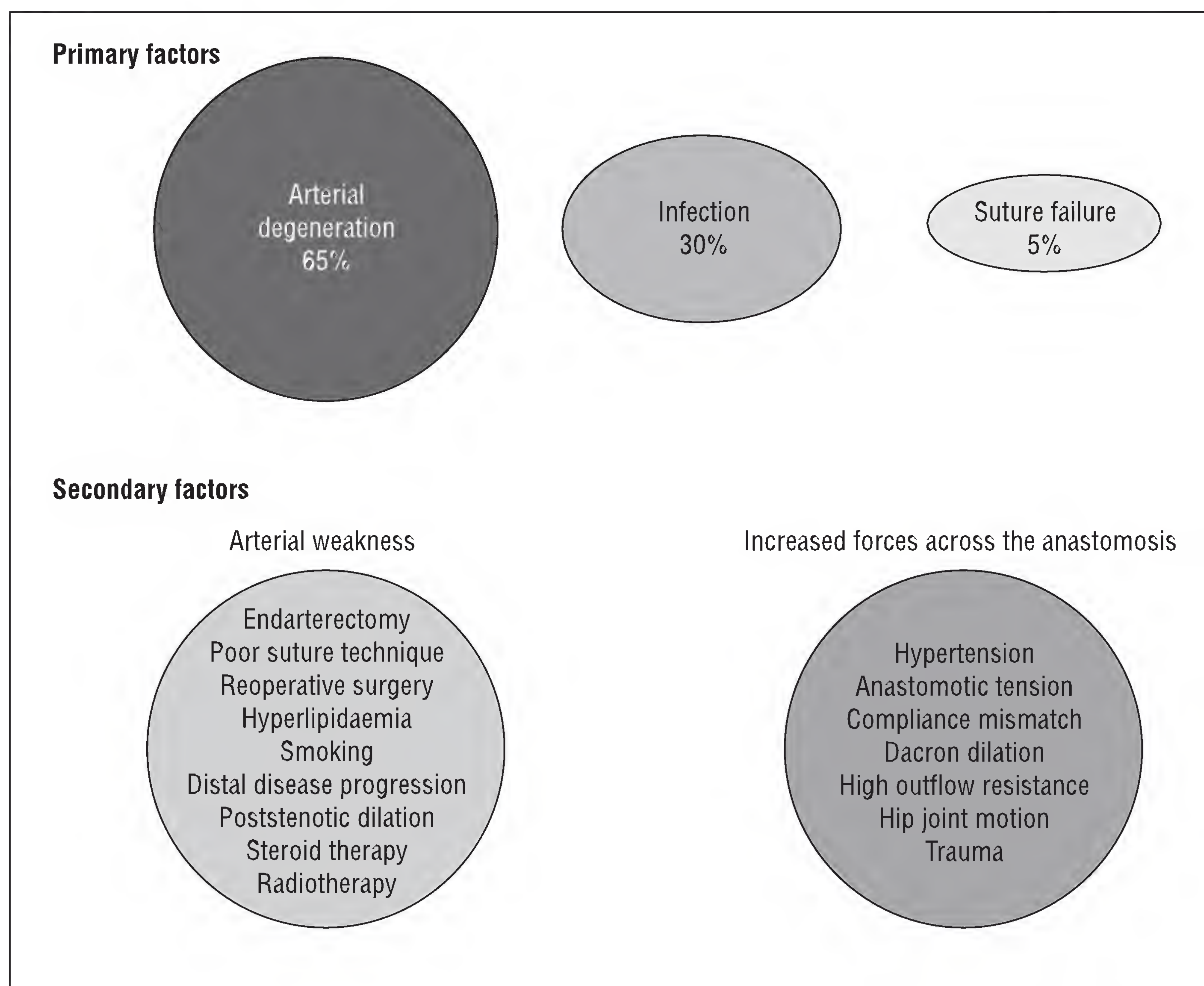


Fig. 9a.2. Aetiology of anastomotic aneurysms.

The aetiology is summarised in Fig. 9a.2; there are three primary factors and a number of secondary factors. One of the first documented causes was suture failure, when braided silk was employed [5]. Since monofilament sutures have been used, suture failure has become a less common factor, although occasionally reported disasters highlight the importance of careful suture handling to avoid cracking [6].

Arterial degeneration is the most common primary factor. The disease process that mandated the bypass continues after its insertion [1, 7, 8]. Histologically, a chronic inflammatory response can be identified at an anastomosis [9].

Secondary factors are numerous and compound the process of arterial degeneration [10]. Poor technique, failing to suture all layers of the artery, use of Dacron, and the need for endarterectomy all weaken the arterial graft complex [1]. Hypertension and high outflow resistance may theoretically increase strains at the anastomosis, together with physical disruption from both hip motion and post-stenotic dilation as the graft passes under the inguinal ligament [9]. These and other factors can cause compliance mismatch, which may also be a factor [8].

Anastomotic aneurysms can be caused by local infection. Infection with high-virulence bacteria, such as *Staphylococcus aureus*, usually presents early with clinical graft infection. Late anastomotic rupture is often caused by low-virulence organisms, such as *Staphylococcus epidermidis*. Up to 30 per cent of anastomotic aneurysms can be shown to harbour pathogenic bacteria at reoperation [7]. This has implications for surgical repair (see below). **[Q2: D, C, B, A, E]**

Indications for Surgical Intervention

Surgery for anastomotic aneurysms is aimed at controlling symptoms or preventing the onset of complications. Symptoms of pain are associated with the enlarging mass or pressure on adjacent structures, such as the femoral nerve. Complications may be local or distal. The enlarging aneurysm may occlude the underlying vessel, causing distal ischaemia. Emboli associated with flow disruption may be propagated distally. Aneurysm rupture represents the greatest worry but is relatively rare.

Complications are related to aneurysm size. Therefore, conservative management may be undertaken if the aneurysm is small and easily accessible, and demonstrates no evidence of progressive enlargement or symptoms. Aneurysms less than 2 cm in diameter can be observed safely [1]. Above this size, the incidence of complications rises and surgical intervention should be considered. However, the medical state of the patient may necessitate selective aneurysms larger than 2 cm being managed conservatively by watchful waiting.

False aneurysms caused iatrogenically following direct arterial puncture must be differentiated from anastomotic aneurysms because their treatment differs substantially. False aneurysms following sterile arterial puncture may be treated by arterial compression under duplex imaging [11]. More recently, injection of thrombin into these false aneurysms has been shown to be safe and effective, even in anticoagulated patients [12]. This technique is not suitable for anastomotic aneurysms. Other radiological techniques may be used selectively for false aneurysms in inaccessible positions, such as the renal or subclavian arteries, where coil embolisation may be used to occlude the feeding vessel [13]. Again, this is rarely suitable for anastomotic aneurysms. Occasionally, it is possible to insert a covered stent across an anastomotic aneurysm to produce local aneurysm thrombosis [14, 15] and to maintain normal distal flow. The ideal indication for this is intra-abdominal aortoiliac anastomotic aneurysms, where reoperation carries substantial risk. It is important that these non-operative techniques are not used in situations where there is any risk that the false aneurysm is due to infection. The most common site for anastomotic aneurysm is the groin, where non-operative techniques have not been found to be effective. The groin is also easily accessible for surgery, so direct operation is the usual intervention in this situation. **[Q4: A, B, C, D] [Q3: A, B, C, D]**

Surgery for Anastomotic Aneurysms

Surgical repair should be undertaken in fit patients with large or symptomatic anastomotic aneurysms. Local repair is usually possible in non-infected aneurysms, although graft replacement may be necessary. If infection is the cause of the aneurysm, then more extensive repairs with ligation and remote bypass or replacement of the entire initial graft may be needed [16].

Anastomotic aneurysms usually occur in arteriopathic patients. Careful preoperative planning is needed to make the patient as fit as possible. General anaesthesia is needed to allow adequate exposure, and the surgery is carried out under antibiotic and heparin cover. Once vascular control above and below the aneurysm has been obtained with minimal dissection, the aneurysm should be opened, along with the entire abnormal artery. Occlusion balloon catheters are often helpful in obtaining vascular control in this situation. The false aneurysm is usually resected and the ends of the graft and artery freshened for reanastomosis. Interposition grafting is

likely to be needed to ensure that the new anastomosis is created without tension. Autologous saphenous vein is the graft of choice, although often polytetrafluoroethylene (PTFE) or Dacron may be better for size matching. **[Q4: E, C, B, A, B]** Retroperitoneal anastomotic aneurysms present more of a challenge. Proximal aortic anastomotic aneurysms may require supracoeliac clamping or balloon occlusion catheters [17]. Aneurysms associated with the distal portion of an aortoiliac graft may present late and catastrophically, illustrating the potential importance of monitoring these grafts for a prolonged period [18].

Infection in Anastomotic Aneurysms

Some 80 per cent of anastomotic aneurysms occur in the groin, and they have the highest incidence of infection as their primary cause; approximately 30 per cent contain pathogenic bacteria. A high level of clinical suspicion of infection must be maintained, and Gram staining of all clots and removed graft should be carried out as a matter of routine. Perioperative antibiotics should be continued until results are available.

The diagnosis of infection is usually obvious if the graft is surrounded by pus. If the graft is frankly infected, it should be excised completely with an extra-anastomotic bypass to restore the distal circulation with prolonged, high-dose antibiotic cover. An obturator bypass may be used for an infected femoral false aneurysm. Aortic stump oversewing and axillobifemoral grafting can treat the (fortunately rare) infected aortic anastomotic aneurysm. Morbidity and mortality rates are high.

Grafts with a more indolent level of infection that becomes apparent only after microbiological investigation may be treated less radically. It is safest to assume that all femoral anastomotic aneurysms are contaminated. If prosthetic material is needed for repair, then measures used to reduce the chance of reinfection include the use of a rifampicin-soaked, gelatine-coated Dacron graft and gentamicin beads laid in close proximity. The reinfection rate after such procedures is 10 per cent [19].

Outcome

Outcome depends on the initial site of the aneurysm and any confounding factors [20]. As the most common site for anastomotic aneurysms, the femoral artery has one of the highest successful outcomes. About 90 per cent of cases will have successful surgery, and those that have a recurrence still have a 90 per cent success rate from a second or subsequent operation. In comparison, anastomotic aneurysms that are intra-abdominal have a high complication rate when repaired surgically. A small anastomotic aneurysm in a superficial position can be monitored by ultrasound initially, and can then be monitored safely by repeated examination by a clinician or the motivated patient. The success rate of operation at these sites is good [21]. Retroperitoneal aneurysms require long-term ultrasound follow-up. **[Q6: F, T, F, T, F]** If possible, minimally invasive techniques should be used for repair to avoid the high morbidity and mortality associated with surgery (providing infection is not present). In patients fit for surgery, excision and graft interposition has excellent long-term results.

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9b. False Aneurysm in the Groin Following Coronary Angioplasty

Steven S. Kang

A 70-year-old female with a history of hypertension developed chest pair and was admitted to a local hospital. Heparin was administered intravenously. Later that day, she underwent coronary angiography, which showed a critical stenosis of the left anterior descending artery. The lesion was treated with angioplasty and stent placement. The right femoral artery sheath was left in place overnight, and heparin was continued. The following morning after stopping heparin, the sheath was removed and a FemoStop device was placed over the groin for 4 h. Heparin was then restarted.

The next day, the patient was without any chest pain, but she did have mild discomfort in the right groin. There was a large hematoma in the right groin. The overlying skin had ecchymosis. The femoral pulse was prominent, and popliteal and pedal pulses were normal. A systolic bruit was heard over the femoral artery.

Question 1

What test should be obtained at this time?

A. Computed tomography scan with intravenous contrast.

B. Duplex ultrasound.

C. Magnetic resonance angiogram.

D. Contrast arteriogram.

A false aneurysm was suspected and confirmed by duplex ultrasound examination. It was arising from the common femoral artery (CFA). The flow cavity measured 3 cm in diameter (Fig. 9b.1).



Fig. 9b.1 Duplex ultrasound demonstrates a false aneurysm arising from the common femoral artery.

Question 2

The incidence of postcatheterization false aneurysms in the groin is higher under which of the following situations?

A. Puncture of the CFA instead of the superficial femoral artery (SFA).

B. Use of larger sheaths.

C. Use of postprocedural anticoagulation.

D. Patients with hypertension.

E. Manual compression versus mechanical compression with a FemoStop after catheter removal.

Question 3

Which of the following statements about postcatheterization false aneurysms is/are true?

A. Urgent surgical repair is indicated.

B. This aneurysm is likely to undergo spontaneous thrombosis if observed.

C. Spontaneous thrombosis is less common in patients who are anticoagulated.

D. They may cause deep venous thrombosis.

Heparin was discontinued and ultrasound-guided compression repair (UGCR) was attempted.

Question 4

Which are the disadvantages of UGCR?

A. Thrombosis of the underlying artery is a frequent complication.

B. Most patients find it painful.

C. It is less successful in patients who are anticoagulated.

D. Approximately 30 percent of successfully thrombosed false aneurysms recur.

Due to patient discomfort, intravenous morphine and midazolam were administered. After 60 min of compression, the false aneurysm still had flow. It was decided not to persist. The hospital did not have any experience with ultrasound-guided thrombin injection. After discussion with our vascular surgery service, the patient was transferred to our hospital for thrombin injection.

Question 5

Which of the following statements regarding ultrasound-guided thrombin injection is/are true?

A. It requires direct injection of thrombin into the neck of the false aneurysm.

B. It involves simultaneous compression of the false aneurysm.

C. It is less painful but less effective than UGCR.

D. It works well in anticoagulated patients.

E. It is appropriate only for femoral false aneurysms.

Thrombin solution (1000 units/ml) was loaded into a small syringe and a 22-gauge spinal needle was attached. Under ultrasound guidance, the needle was placed into the center of the false aneurysm (Fig. 9b.2) and 0.3 ml thrombin was injected slowly. Within 15 s, the false aneurysm was thrombosed completely (Fig. 9b.3). The procedure was tolerated well. Flow in the underlying artery was preserved and pedal pulses were intact. As the patient was otherwise stable, she was discharged soon afterwards.

Question 6

What are the reported complications of thrombin injection?

A. Anaphylaxis.

B. Intra-arterial thrombosis.



Fig. 9b.2. The tip of the needle is visible within the false aneurysm cavity.

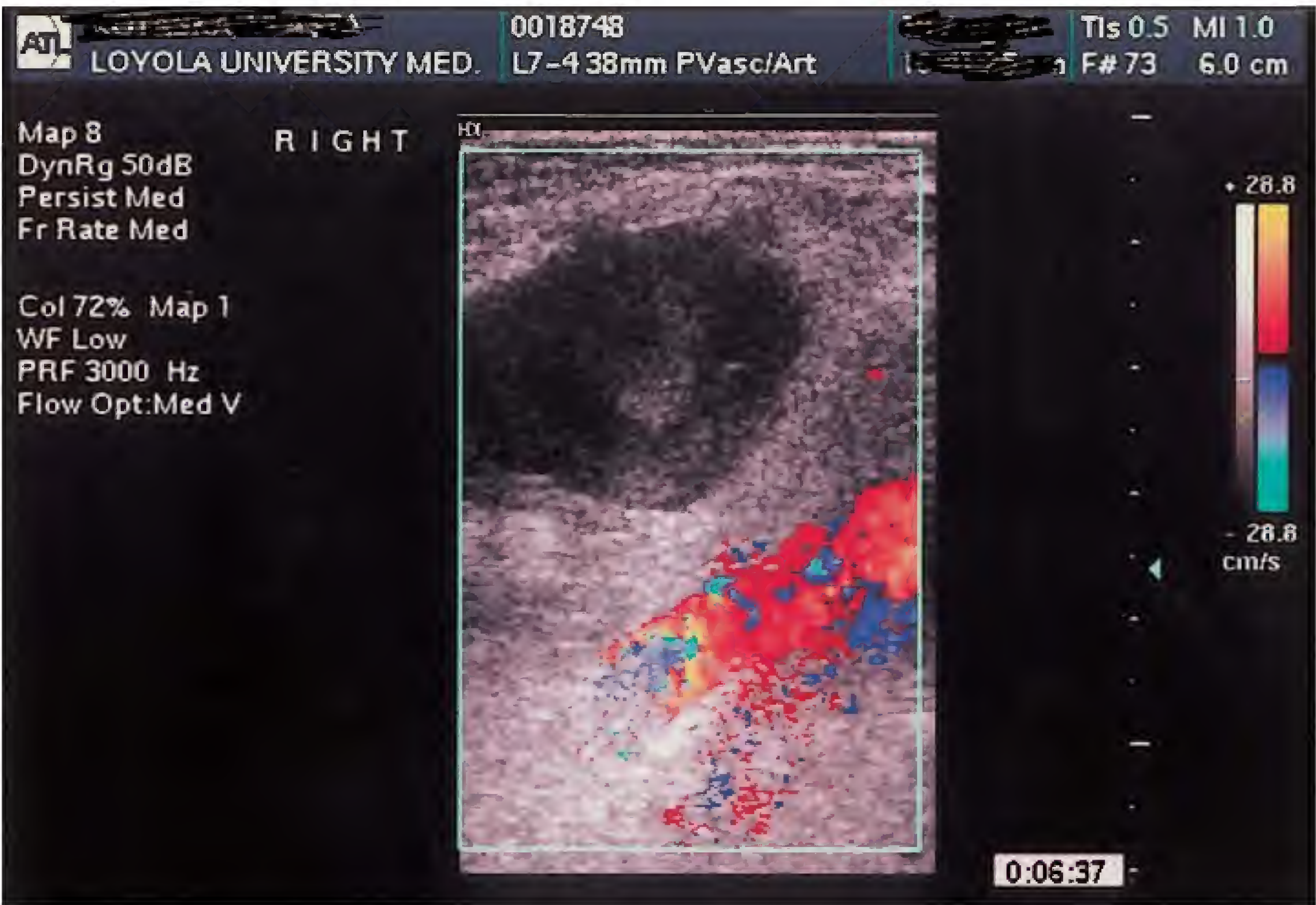


Fig. 9b.3. The aneurysm is completely thrombosed 15 seconds after thrombin injection.

C. Prolonged urticaria.

D. Mad cow disease.

Commentary

A false aneurysm after catheterization is suspected when there is a hematoma, especially an enlarging one, at the puncture site hours or days after the procedure. There is often significant ecchymosis of the overlying skin. There may be a bruit, but a continuous bruit is usually associated with an arteriovenous fistula. There may be pain or neuralgia, and the site is often tender. A pulsatile mass is usually palpable, but a simple hematoma overlying the artery may give the same impression. Only a minority of false aneurysms are diagnosed unequivocally by physical examination. The diagnosis of a femoral false aneurysm has become very easy with duplex ultrasound. **[Q1: B]**

The incidence of postcatheterization femoral false aneurysms varies from less than 0.5 percent to more than 5 percent [1]. Some of the factors that increase the likelihood of false aneurysm formation include larger sheaths, longer procedure times, multiple catheter exchanges, and peri- and postprocedure anticoagulation. Puncture of the superficial femoral or deep femoral artery instead of the CFA is found to be associated with higher rates of false aneurysm formation. Direct manual compression after catheter removal is better than compression devices, such as the FemoStop or C-clamp. Patient characteristics that may increase the likelihood of false aneurysm formation include atherosclerosis of the punctured artery, obesity and hypertension. **[Q2: B, C, D]**

The potential complications of untreated false aneurysms are well known. Rupture is the most dramatic and life-threatening complication. Compression of surrounding tissues can cause pain, neuropathy, venous thrombosis, and necrosis of the overlying skin. Thrombosis of, or embolization into, the femoral artery may occur. Infection of these false aneurysms is less common. Because of these potential outcomes, early surgical repair had been advocated in the past. However, in the 1990s, several series showed that the majority of small false aneurysms will develop spontaneous thrombosis [2–4]. It is less likely to occur with larger false aneurysms or in patients who are on anticoagulants. **[Q3: C, D]** Thrombosis may occur within days, or it may take weeks. Once thrombosis occurs, the false aneurysm is then a simple hematoma that gets resorbed slowly over time. The defect in the artery heals uneventfully in most cases.

In 1991, Fellmeth et al. [5] described the method of UGCR of postcatheterization femoral false aneurysms and arteriovenous fistulas. The ultrasound transducer is used to apply downward pressure on the neck of the false aneurysm to arrest flow. Pressure is maintained until the blood in the aneurysm becomes thrombosed. After the introduction of UGCR, numerous reports were published verifying the efficacy and overall safety of this procedure [6–9]. The typical success rate was between 60 and 90 percent. There were only a few published complications, including thrombosis of the underlying artery or the femoral vein from the compression, rupture during compression, rupture after successful compression, skin necrosis caused by prolonged pressure on the skin, and vasovagal reactions. Therefore, UGCR was shown to be a good alternative to

surgical repair or observation, and most centers made it the initial treatment method.

There are several disadvantages to the procedure. It is time-consuming, requiring an average of 30–60 min of compression. In most hands, the results are significantly poorer for patients on anticoagulants [10]. The recurrence rate is about 4–11 percent, but it is as high as 20 percent for anticoagulated patients [6]. About 10 percent of patients cannot be treated with UGCR because they have false aneurysms that are not compressible or cannot be compressed without also collapsing the underlying artery, which would increase the chance of arterial thrombosis. For most patients, the compression is painful, and intravenous sedation or analgesia is often necessary. Some patients have required epidural or general anesthesia to allow compression. Applying compression is also very uncomfortable for the operator. **[Q4: B, C]**

Various endovascular treatments have been described for false aneurysms that have failed compression. They usually require catheterization of the feeding artery or false aneurysm from a remote access site. Embolization coils can be used to occlude the neck or to fill the cavity of the false aneurysm [11, 12]. Stent grafts can be placed in the femoral artery to exclude the false aneurysm, but late occlusion of the grafts is not uncommon [13]. They certainly should not be the initial method of treatment. However, for false aneurysms arising from other, less easily accessible arteries, these techniques may have a role.

Because of the shortcomings of UGCR, we developed a new method of treating false aneurysms with ultrasound-guided thrombin injection [14, 15]. Thrombin causes the cleavage of fibrinogen into fibrin, which then polymerizes into a solid. It is the final product of the coagulation cascade, and this reaction occurs naturally whenever blood clots. Thrombin has been used topically for many years to control surface bleeding in the operating room. Our technique is as follows: The ultrasound transducer is centered over the false aneurysm. Thrombin at a concentration of 1000 U/ml is placed into a small syringe, and a 22-gauge spinal needle is attached. The needle is inserted at an angle into the false aneurysm along the same plane as the transducer, and the tip is positioned near the center of the false aneurysm. About 0.5 ml thrombin solution is injected slowly into the false aneurysm. Within seconds, thrombosis of the false aneurysm is seen. The procedure is not painful, and patients do not require any analgesia or sedation. We allow patients to get out of bed immediately after treatment, and outpatients are sent home soon after the procedure.

So far, we have had great success with this procedure. We have treated 165 false aneurysms. Most (149) developed after groin puncture. There were also false aneurysms in six brachial, three subclavian, two radial, two tibial, one distal SFA, and one superficial temporal arteries, and in one arm arteriovenous fistula. Forty-seven patients were anticoagulated at the time of thrombin injection. It was initially successful in 161 of 165 patients. The other four (all femoral) had partial thrombosis. One of these had complete thrombosis 3 days later when brought back for repeat injection. Three had surgical repair. There were early recurrences in 12 patients who had initial successful thrombin injection. Seven were reinjected successfully at the time the recurrence was diagnosed. One had spontaneous thrombosis several days after recurrence was identified. Four had surgical repair. Overall, only 7 of 165 required surgical repair. There were three complications. A brachial artery false aneurysm had injection of thrombin directly into its neck, which caused thrombosis of the brachial artery. A femoral false aneurysm had a relatively large volume of thrombin injected and developed a thrombus in the posterior tibial

Table 9b.1. Results of ultrasound-guided thrombin injection

	Cases	Successes (percent)	Complications
Current	165	158 (96)	3
Khoury [18]	131	126 (96)	3
Paulson [19]	114	110 (96)	4
Maleux [20]	101	99 (98)	0
Mohler [21]	91	89 (98)	1
La Perna [22]	70	66 (94)	0
TOTAL	672	648 (96)	11 (1.6)

artery. Both of these thromboses resolved after intravenous heparin. A femoral false aneurysm with a short neck that was about 10 mm wide had partial thrombosis of the aneurysm. Further injection was not able to thrombose the remaining cavity but instead caused a tail of thrombus to form in the SFA. The patient underwent surgical thrombectomy and repair of the aneurysm. **[Q5: D]**

Our results show that intra-arterial thrombosis after thrombin injection is uncommon. The high concentration of thrombin results in almost immediate conversion of the solution into a solid (thrombus) when it mixes with relatively stagnant blood. Since the neck of the false aneurysm is usually much narrower than the aneurysm cavity, the thrombus cannot enter the artery. As long as the volume of the thrombin injected does not approach or exceed the volume of the false aneurysm, which may result in forcing some of the solution out of the cavity, then the risk of native artery thrombosis should be small. It is likely to be higher when the neck is very wide. Other complications that have been reported include single cases of anaphylaxis [16] and prolonged urticaria [17]. Repeated exposure to bovine thrombin can also lead to development of antibodies to bovine factor V, which may cross-react with autogenous factor V, causing hemorrhagic complications. **[Q6: A, B, C]**

Many others have also had good results with this procedure. In the largest series, the success rate is around 96 percent and the complication rate less than 2 percent (Table 9b.1). Given its simplicity, efficacy, and safety, ultrasound-guided thrombin injection should be considered the initial treatment of choice for postcatheterization false aneurysms.

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10. Acute Thrombosis

Vikram S. Kashyap and Kenneth Ouriel

A 60-year-old retired nurse presents to the emergency department with a cold and painful right foot. She has an extensive history of peripheral vascular disease including bilateral iliac stenting for atherosclerotic occlusive disease. Prior to the onset of symptoms, she could walk at least a quarter of a mile without pain. Her past history includes a stroke years ago that she has recovered from, hyperlipidemia, coronary artery disease, and smoking. She has not been recently hospitalized or undergone any invasive procedure or operation.

On examination, her pulse is 90 bpm, and her blood pressure is 115/65. Her heart sounds reveal a regular rhythm. She has a normal left femoral pulse and the left foot is warm and well perfused. The right limb has no palpable pulses and the right foot is pale and cool. She can move the foot, but the toes are insensate. There is a venous Doppler signal in the right foot, but no arterial signal.

Question 1

Native arterial or graft thrombosis can be differentiated from embolic occlusion by the following:

A. The presence of palpable pulses in the contralateral extremity.

B. A history of cardiac arrhythmias.

C. The degree of profound ischemia in the affected extremity.

D. The location of the occlusion.

E. All of the above.

Question 2

What is the SVS/ISCVS category of limb ischemia in this patient?

A. Category I.

B. Category IIa.

C. Category IIb.

D. Category III.

Question 3

In acute embolism, the sequence of events is:

A. Paralysis, pain, paresthesia, pulselessness, pallor.

B. Pulselessness, pain, pallor, paresthesia, paralysis.

C. Pulselessness, pain, pallor, paralysis, paresthesia.

The patient is taken to the endovascular suite and an angiogram is performed via a contralateral femoral approach. This reveals an occluded right iliac stent (Fig. 10.1)



Fig. 10.1. Aortography via a left femoral approach documents a right iliac thrombosis in the setting of prior iliac stenting.



Fig. 10.2. Arteriography of the right leg documents reconstitution of the common femoral artery with a normal run-off via both anterior and posterior tibial arteries (not shown).

with reconstitution of the femoral bifurcation (Fig. 10.2) and normal outflow to the foot.

Question 4

Treatment options for this patient include:

A. Anticoagulation with heparin and coumadin.

B. Operative thrombectomy.

C. Extra-anatomic bypass.

D. Aortofemoral bypass.

E. Mechanical thrombectomy, thrombolysis, and endovascular intervention.

F. Intravenous thrombolysis.

Question 5

After thrombolysis, long-term outcome is predicated on:

A. The thrombolytic agent used.

B. Unmasking a "culprit lesion" that is treated via either endovascular or surgical means.

C. The length of thrombolysis.

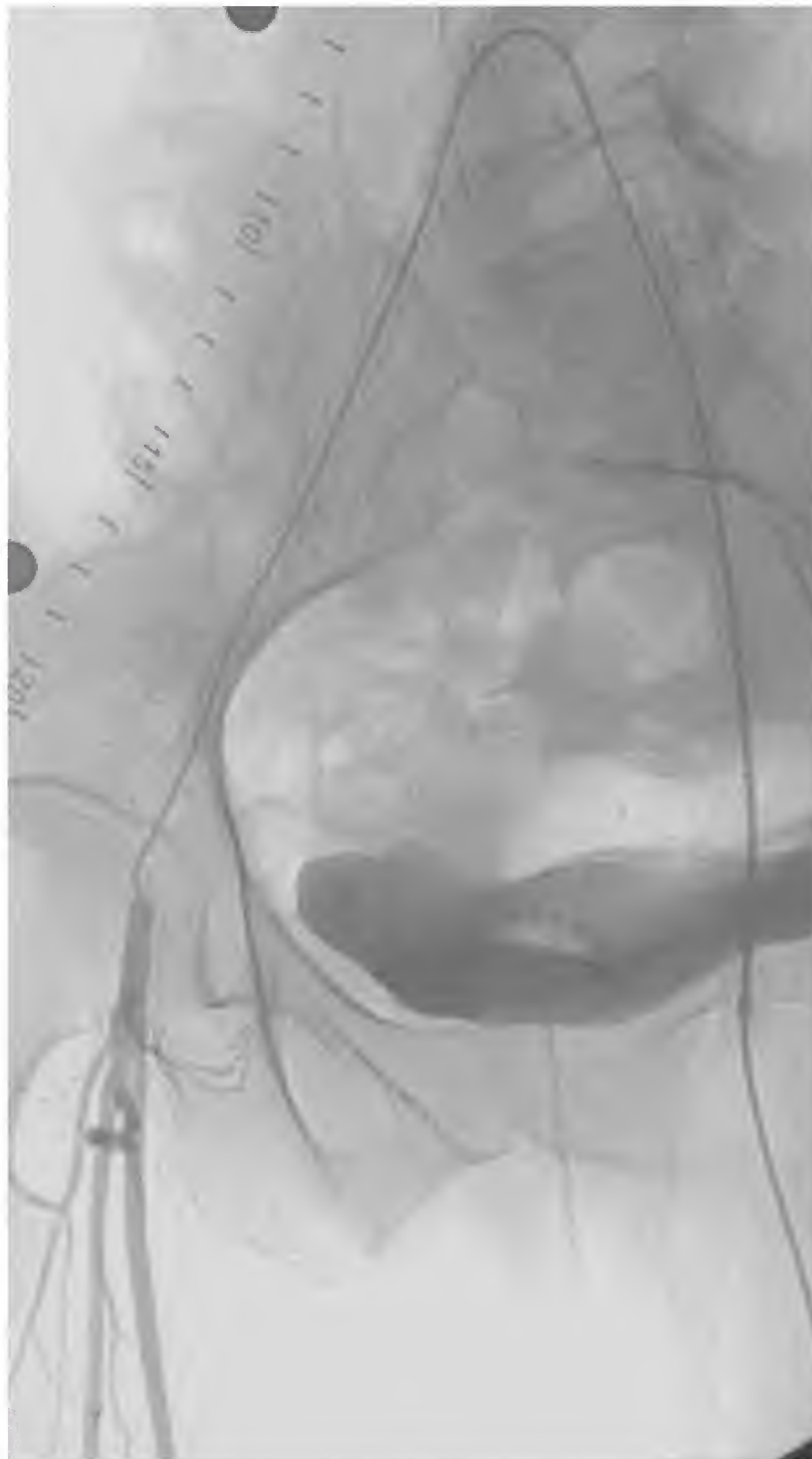


Fig. 10.3. A hydrophilic wire is used to safely cross the thrombosed segment and this arteriogram confirms catheter placement in the native circulation distal to the iliac thrombosis.

D. Assuring all acute thrombus is lysed.

E. The arterial outflow.

This patient underwent successful thrombolysis. A combination of a hydrophilic wire and catheter was used to cross the acute thrombotic occlusion ("guidewire traversal test") and gain access to the native femoral system (Fig. 10.3). Thrombolysis (agent: alfimeprase, Nuvelo, Inc., investigational use only) was performed through a multi-side hole infusion catheter and at 2 hours after initiation, significant thrombus resolution had occurred (Fig. 10.4). The patient was monitored closely and returned to the endovascular suite for a follow-up study.

Contrast injection through the sheath demonstrated a stenotic lesion and percutaneous balloon angioplasty of the distal right iliac system was performed (Fig. 10.5) with restoration of blood flow into the right limb without any evidence of distal embolization. The patient had palpable femoral, popliteal, and pedal pulses at the completion of the procedure. She was carefully observed for the development of compartment syndrome. She was discharged 3 days after admission with

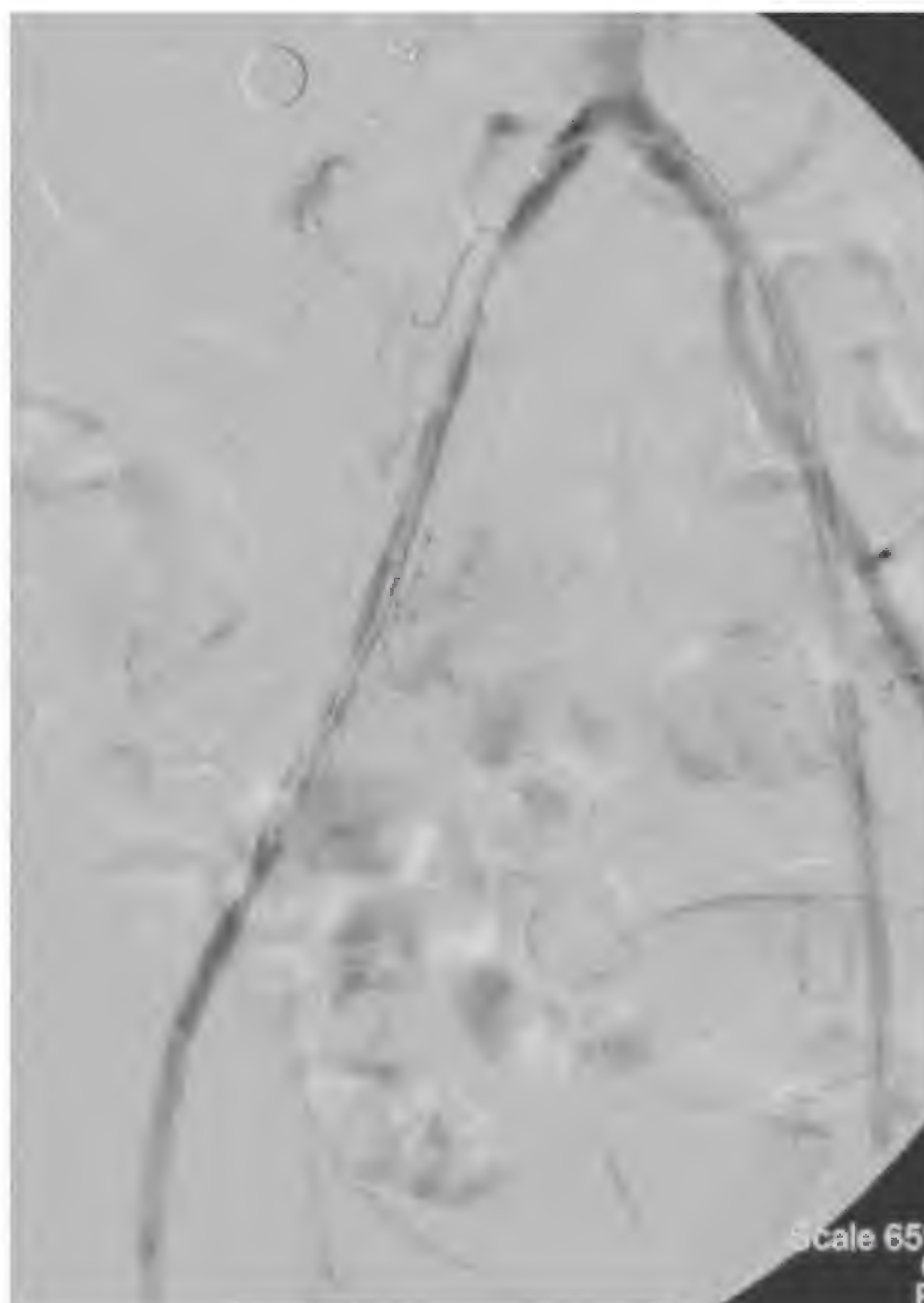


Fig. 10.4. After 2 hours of thrombolysis (alfimeprase, Nuvelo, Inc., investigational use only), approximately 50 percent of the thrombus burden is dissolved.



Fig. 10.5. After complete thrombolysis, a distal iliac stenosis is uncovered and treated with balloon angioplasty. This lesion was presumably the etiology of the acute thrombosis.

an ankle-brachial index of 0.9. The patient has remained free of symptoms for over 6 months.

Commentary

The etiology of acute limb ischemia can be classified into two groups. Thrombotic events occur in the setting of native arterial disease or bypass graft stenoses. In contrast, embolic phenomena usually occur in normal vessels and tend to lodge at arterial bifurcations [1]. Thrombotic occlusions are thought to represent progression of atherosclerotic disease and occur at sites along the arterial tree and most notably the superficial femoral artery (SFA) at the adductor canal. In comparison, autologous grafts fail at sites of intimal hyperplasia, or fibrotic valves. Due to preexisting

collaterals, native arterial thrombosis seldom presents with the profound ischemia seen with embolic ischemia. The presence of palpable pulses on the contralateral limb and a history of cardiac arrhythmia assist in differentiating acute embolus from thrombotic occlusions [1].

All of the factors listed can help in differentiating embolic from thrombotic occlusions. **[Q1: E]** Often, a definitive diagnosis cannot be made preoperatively. However, identifying an embolic source for acute limb ischemia is helpful for both the acute and long-term management of the patient.

Clinical classification and diagnosis of acute occlusion of the lower extremity is based on the symptoms of the patient. The severity of symptoms is associated with the extent of the occlusion and the presence of preexisting vessels. Patients with thrombotic occlusions from underlying disease of the SFA at the adductor canal may only experience worsening claudication while embolic events are usually associated with rapid onset and severe ischemia because of the lack of preexisting collateral flow. Limb ischemia has been classified into three categories by an SVS/ISCVS ad hoc committee, based on severity of ischemia [2]. Category I limbs are viable, not immediately threatened and have no motor or sensory loss. There are clearly audible arterial Doppler signals in the foot. Category II includes threatened limbs where salvage may be possible with timely intervention. Importantly, this category is divided into two subgroups, a and b, which distinguish the time interval necessary for treatment. Category IIa patients require prompt treatment whereas category IIb patients need immediate therapy to prevent amputation. In category II, audible venous Doppler signals are present, but there is no arterial signal in the foot. Category IIa patients have minimal sensory loss and have no motor loss. However, patients with category IIb ischemia have muscle weakness, and sensory loss encompasses more than the toes. Category III is characterized by irreversible ischemia with profound and permanent neuromuscular damage where amputation is the only recourse.

This patient has category IIa limb ischemia characterized by mild sensory loss and lack of distal arterial signals in the foot. **[Q2: B]**

The sequence of clinical events in patients with lower extremity ischemia is often predictable. **[Q3: B]** Most patients with acute ischemia, especially of an embolic nature, will have pulselessness followed by pain and pallor. Paresthesia indicates sensory nerve ischemia and occurs usually from 1 to 3 hours after the onset of acute ischemia. Paralysis indicates motor nerve damage that is often irreversible. In the setting of acute ischemia without collateral flow, paralysis occurs approximately 6 hours after the onset of ischemia [3]. Any motor dysfunction should be seen as a worrisome sign and should prompt urgent intervention. Poikilothermia indicates that the foot or limb has approximated ambient temperature. In these irreversible cases (category III), amputation is the only option and often has to be done quickly to avoid systemic complications.

Both the diagnosis and localization of acute arterial occlusion is based upon the findings on physical examination and imaging studies. A "waterhammer" pulse signifies outflow obstruction, as observed with a common femoral embolus. By contrast, calcified vessels are common with thrombosis from underlying atherosclerotic disease. Multiple options are available for localization of the occlusion. Noninvasive testing with segmental pressures, pulse volume recording, and measurement of the ankle brachial indices can provide a baseline study for comparison after treatment. Both vertical and horizontal pressure gradients of 30 mm Hg or more in the lower extremity can accurately identify the site of occlusion. Duplex ultrasonography can

also be utilized to examine the femoral and popliteal vessels and localize area of occlusion. Other causes such as thrombosed popliteal aneurysm can be easily diagnosed in this manner. Magnetic resonance angiography and CT angiography are emerging as noninvasive techniques for arteriographic imaging and localization of thrombosis. However, angiography remains the gold standard for localization of arterial occlusion. As importantly, angiography allows percutaneous access to the site of thrombosis and an array of treatment options for restoring blood flow to the limb.

Treatment for limb ischemia has evolved over the past two decades with advances in both pharmacologic therapy and endovascular options. **[Q4: B, C, E]** In patients with acute limb ischemia secondary to iliac occlusion, operative thrombectomy of the occluded iliac system may be feasible. In the setting of profound ischemia and a diseased iliac artery precluding successful thrombectomy, extra-anatomic bypass can be performed to provide expeditious blood flow into the ischemic limb. In these cases, either femoral-femoral bypass or axillo-femoral bypass can be contemplated depending on the inflow source. Aortofemoral bypass is a very durable option for patients with chronic occlusion and chronic ischemia of the limb. However, in patients with acute ischemia ill-prepared for major surgery, proceeding with direct reconstruction with the aorta as the inflow is sometimes hazardous. Multiple endovascular devices are available in the setting of acute thrombosis. Percutaneous mechanical thrombectomy with thrombolysis either via the power-pulse technique or via a standard infusion often quickly resolves the acute ischemia. Continued thrombolytic infusion is required for complete resolution of thrombus. Often, a "culprit" lesion will be unmasked by dissolving all of the acute thrombus, allowing percutaneous treatment of the offending lesion. Systemic thrombolytic therapy has been used to treat peripheral arterial occlusions, but results have been disappointing owing to a significant incidence of bleeding complications. Currently, systemic therapy is usually used for venous thromboembolic states. Regional intravascular infusion of the lytic agent avoids some of the systemic complications and is largely used for peripheral arterial thromboses and graft occlusions. Because a systemic lytic state may occur with prolonged regional intravascular thrombolytic therapy, patient selection is critical. Absolute contraindications include active internal bleeding, recent surgery or trauma to the area to be perfused, recent cerebrovascular accident, or documented left heart thrombus [3]. Relative contraindications include recent surgery, gastrointestinal bleeding or trauma, severe hypertension, mitral valve disease, endocarditis, hemostatic defects, or pregnancy.

Several multicenter trials have examined groups of patients treated with surgical therapy or thrombolysis. **[Q5: B, D, E]** The Rochester trial randomized patients to surgery or thrombolysis and demonstrated a lower mortality in the thrombolysis group [4]. Following successful thrombolysis, unmasked "culprit lesions" were treated with angioplasty or surgery of a lesser magnitude, thereby reducing the severity of the intervention and overall morbidity. The finding of a lesion that precipitated the thrombosis is critical to avoiding re-thrombosis. The STILE trial (Surgery versus Thrombolysis for Ischemia of the Lower Extremity) compared optimal surgical therapy to intra-arterial catheter-directed thrombolysis for native arterial or bypass graft occlusions [5, 6]. Stratification by duration of ischemic symptoms revealed that patients with ischemia of less than 14 days' duration had lower amputation rates with thrombolysis and shorter hospital stays, while patients with ischemia for longer than 14 days who were treated surgically had less ongoing or recurrent ischemia and trends toward lower morbidity. At 6 months, amputa-

tion-free survival was improved in patients with acute ischemia treated with thrombolysis, but patients with chronic ischemia had lower amputation rates when treated surgically. Fifty-five percent of patients treated with thrombolysis had a reduction in magnitude of their surgical procedure. Of note, no difference was seen between the use of rt-PA and urokinase [5].

A multicenter, randomized, prospective trial comparing thrombolysis to surgery for acute lower extremity ischemia of less than 14 days' duration has been carried out [7]. The Thrombolysis or Peripheral Arterial Surgery trial (TOPAS) randomized 757 patients to surgery or thrombolytic therapy. The most effective dose for urokinase was determined to be 4000 U/min with complete thrombolysis in 71 percent of patients. After successful thrombolytic therapy, either surgical or endovascular intervention was performed on the lesion responsible for the occlusion if found. When compared to the surgical arm, the one-year limb salvage rates and mortality were not statistically different. However, although no statistical differences between the two groups were seen with respect to amputation-free survival, thrombolysis was associated with a reduction in the number and magnitude of open surgical interventions over a one year follow-up period.

Unlike thrombolysis in coronary or venous systems, dissolution of the larger peripheral arterial thrombi requires direct infusion of thrombolytic agent into the clot. The thrombosed artery or bypass graft must be accessed with a wire, followed by placement of an infusion system into the thrombus. Following successful thrombolysis, any unmasked lesion can be addressed with balloon angioplasty and stenting or with an open surgical procedure. Even when a surgical procedure is necessary, it can usually be performed electively, in a well-prepared patient, and is often of a lesser magnitude than what would have been required without thrombolysis. Thrombolytic therapy is an effective option for selected patients with acute thrombotic occlusion.

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11. Arterial Embolism

Andre Nevelsteen

A 65-year-old man presented with acute severe pain in his right leg. Medical history revealed non-insulin-dependent diabetes mellitus for 3 years and a myocardial infarction (MI) some 5 years ago. The pain in the right leg developed suddenly over 6 hours without associated trauma and became worse over time. On admission, the right leg looked pale distally from the level of the knee. There was loss of light touch sensation on examination of the foot. The patient had difficulties in wiggling the toes. Plantarflexion and dorsiflexion of the toes were still possible. Palpation of the calf showed soft but tender muscles. Clinical examination of the abdomen showed no abnormalities. There was no pulsating mass. Irregular but bounding pulsations were felt in the right femoral artery. Popliteal artery and tibial artery pulsations were absent. Normal pulsations were felt in the left popliteal and posterior tibial artery.

Question 1

What is the aetiology of arterial embolism?

A. The aetiology of arterial embolism is most frequently unknown.

B. The most frequent cause of arterial embolism is cardiac valve destruction by rheumatic heart disease or endocarditis.

C. The most frequent cause of arterial embolism is atrial fibrillation in association with atherosclerotic heart disease.

D. Deep venous thrombosis might represent a rare cause of arterial embolism.

E. Arterial embolism is most frequently seen in the presence of increased blood viscosity.

With the diagnosis of acute arterial ischaemia in mind, a full dose of intravenous heparin was administered immediately.

Question 2

What is the place of heparin in the treatment of arterial embolism?

A. Heparin can dissolve an arterial embolus, avoiding the need for subsequent operation.

B. Heparin will avoid subsequent arterial thrombosis, which can complicate treatment of arterial embolism.

C. Heparin will avoid subsequent arterial thrombosis, which can complicate treatment of arterial embolism. In addition, heparin will prevent recurrent emboli.

D. The use of heparin is contraindicated since it may lead to fragmentation of an arterial embolism and induce microembolisation in the peripheral arteries.

A chest film X-ray showed no abnormalities. Electrocardiogram (ECG) revealed atrial fibrillation and signs of an old MI. Laboratory studies were normal. Duplex examination showed a thrombotic occlusion of the right femoral bifurcation and the superficial femoral artery. A weak flow sign was present in the popliteal artery. The tibial arteries were not visualised.

Question 3

The preferred treatment of arterial embolism is:

A. Local excision of the vessel and reconstruction with interposition graft.

B. Continued heparinisation and wait and see.

C. Simple Fogarty catheter embolectomy with peroperative angiographic control.

D. Simple Fogarty catheter embolectomy, but percutaneous aspiration thromboembolectomy might be a good alternative in selected cases.

After placement of a central venous catheter, the patient was taken to the operating theatre and the right femoral bifurcation was exposed under local anaesthesia. A transverse arteriotomy confirmed complete thrombotic occlusion of the femoral bifurcation. There was good inflow. Thrombi were removed from the femoral bifurcation, and pulsatile backflow was obtained from the profunda femoris artery.

Multiple thrombi were removed from the superficial femoral artery and the popliteal artery after several passages of Fogarty embolectomy catheters numbers 3 and 4. Intraoperative angiography showed good patency of the superficial, popliteal and peroneal arteries. The anterior tibial artery was completely occluded. The posterior tibial was patent in its first portion but occluded distally. A small catheter was inserted into the popliteal artery, and 350,000 units of urokinase were infused as a dripping infusion over 30 min. Repeated angiography showed further clearance of the posterior tibial artery to the level of the ankle joint. The anterior tibial artery was still occluded. It was decided to accept the situation. The arteries were flushed with a diluted heparinised saline solution, and the transverse arteriotomy was closed with the aid of a Dacron patch. Sodium bicarbonate was administered intravenously before reperfusion.

Question 4

Reperfusion syndrome after arterial embolectomy:

A. Will never be seen after peripheral but only after aortic embolism.

B. Cannot be prevented medically.

C. Will be prevented by early ambulation.

D. Is induced by metabolic acidosis and myoglobinuria.

Postoperatively, the foot was well vascularised and the patient was able to wiggle his toes almost normally. Pulsations were felt in the posterior tibial artery. Intravenous heparin was continued. Brisk diuresis was maintained with mannitol and alkalisation of the urine. Repeated laboratory studies showed no evidence of acidosis or hyperkalaemia.

Question 5

Fasciotomy:

A. Has become obsolete and swelling of the limb should be treated by elevation and bed rest.

B. Is best routinely performed in any patient, treated for arterial embolism of the lower limbs.

C. The indication to fasciotomy needs to be based on objective parameters such as the presence of reperfusion syndrome and postoperative compartmental pressure measurements.

D. In daily practice, the indication for fasciotomy is most frequently based on individual preference and clinical feeling.

Six hours postoperatively, the patient developed significant limb swelling with augmentation of pain, venous hypertension and sensory impairment of the foot. A perifibular fasciotomy to decompress all four compartments was performed under general anaesthesia. Afterwards, the swelling subsided and the fasciotomy wound was closed in a delayed primary fashion after 1 week.

Question 6

With the pre- and peroperative diagnosis in mind:

A. The patient should be placed under antiplatelet therapy postoperatively in order to prevent another episode of embolism.

B. Heparin and oral anticoagulants remain the treatment of choice during the postoperative period.

C. Subsequent investigation with regard to the source of the embolus is not necessary, because this will not change the medical treatment.

D. Postoperative investigation with regard to the source of embolism can be limited to cardiac examinations such as echocardiography and Holter monitoring.

Abdominal ultrasound performed postoperatively showed atheromatosis of the abdominal aorta but no aneurysmal dilatation. Transthoracic and transoesophageal echocardiography revealed no ventricular aneurysm or intracardiac thrombi. Holter monitoring for 24 h confirmed atrial fibrillation. Pathological examination of the retrieved emboli was compatible with ordinary thrombotic material. Cultures were negative. The problem of atrial fibrillation was handled medically. Oral anticoagulation was initiated, and the patient was discharged after 10 days. Six months later, there were no repeat episodes of acute ischaemia.

Commentary

Acute ischaemia due to arterial embolism represents a limb-threatening event. Although the carotid or intracranial vessels may be involved in a minority of the cases, the upper or lower extremities are involved in 70–80 per cent in most series of arterial embolisation [1]. The lower extremity is involved five times as frequently as the upper extremity, and the sites of embolic occlusion are most often related to major arterial bifurcations. The common femoral bifurcation is the most frequent site of embolic occlusion, usually noted in 30–50 per cent of all cases [2]. In total, the femoral and popliteal arteries are involved more than twice as often as the aorta.

The heart is by far the predominant source of arterial emboli, seen in 80–90 per cent of cases [3]. Atrial fibrillation is present in approximately 70 per cent of patients. Previously, it was most frequently the reflection of rheumatic heart disease. Since the incidence of rheumatic heart disease has declined steadily over the last 50 years, atrial fibrillation is now associated most frequently with atherosclerotic heart disease.

MI is the second common cause of peripheral embolisation. Left ventricular mural thrombus occurs in 30 per cent of acute transmural infarcts. Clinically evident embolism is seen in only 5 per cent of these patients [4]. One should be aware, however, that silent MI may be present in up to 10 per cent of patients with peripheral emboli, and that embolisation may be the presenting symptom of an acute infarction. Apart from the acute period, MI may also cause emboli after longer intervals. This is usually due to areas of hypokinesis or ventricular aneurysm formation. Although most emboli occur within 6 weeks of MI, much longer intervals may be noted.

Other cardiac diseases are associated less frequently with peripheral emboli. Thromboemboli can, however, arise from prosthetic cardiac valves or from vegetations on the mitral or aortic valve leaflets. Endocarditis should certainly be ruled out. Finally, intracardiac tumours, such as atrial myxoma, may also give rise to clinically evident embolic events.

Non-cardiac sources of peripheral emboli are noted less frequently. Major emboli may arise from aneurysms of the aorta or less frequently from the femoropopliteal vessels [5]. With upper-extremity emboli, one should be aware of unsuspected tho-

racic outlet syndrome and aneurysmal deformation of the subclavian artery. Paradoxical emboli might be seen with deep venous thrombosis in association with a patent foramen ovale. Primary or secondary lung tumours might invade the pulmonary veins, causing tumour emboli. Finally, apart from rare causes such as foreign body embolisation, it should be recognised that the source of embolisation will remain inapparent in some 10 per cent of patients [2]. **[Q1: C, D]**

The diagnosis of acute ischaemia caused by arterial embolism is usually straightforward. The most typical signs are characterised by the “five Ps”: pulselessness, pain, pallor, paraesthesia and paralysis. The level of occlusion is determined by the presence or absence of palpable pulses. Once the diagnosis of acute arterial ischaemia has been made, 5000 units of heparin are administered intravenously. This is not meant as effective treatment but it prevents the propagation and fragmentation of the thrombus. Concomitant venous thrombosis, which can occur with prolonged severe arterial ischaemia, might also be avoided. Heparin administration allows time for diagnosis, evaluation and, if necessary, treatment of cardiac disturbances. **[Q2: B]**

Fogarty catheter embolectomy remains the treatment of choice in most patients with peripheral embolisation [6]. The procedure is usually carried out under local anaesthesia and is effective in cases of major emboli. All retrieved emboli should be sent for pathological and microbiological examination. The operative result should be checked by intraoperative fluoroscopy or angioscopy. Remaining thrombi in the distal vessels can be approached directly or by intraoperative thrombolysis [7]. Thrombolytic therapy or percutaneous aspiration thromboembolectomy (Fig. 11.1) may be used as alternatives to Fogarty catheter embolectomy in selected cases with no motor dysfunction or profound sensory loss [8, 9]. **[Q3: C, D]**

All patients undergoing revascularisation of an acutely ischaemic limb are at risk of ischaemia reperfusion syndrome. This was first emphasised by Haimovici [10], described under its most grave form as the myonephropathic-metabolic syndrome. This reperfusion syndrome is the consequence of muscular hypoxia and the associated metabolic changes. A prolonged period of ischaemia results in accumulation of potassium, lactic acid, myoglobin and other cellular enzymes, leading to a significant fall in blood pH due to anaerobic metabolism, paralysis of the sodium potassium cellular pump and rhabdomyolysis [11]. Acute washout of these products may lead to hyperkalaemia and metabolic acidosis, resulting in myocardial depression or dysrhythmias. Myoglobin and other products of skeletal muscle breakdown can precipitate within the kidney and result in acute renal failure. Myoglobinuria is the first sign. **[Q4: D]** These problems should be anticipated with bicarbonate and/or calcium intravenously just before reperfusion. Induction of forced diuresis with mannitol and alkalisation of the urine might avoid acute renal failure. In addition, mannitol also acts as a scavenger of oxygen-derived free radicals, which are an important intermediary in ischaemia reperfusion injury [12, 13]. It is clear, therefore, that the patient should be monitored carefully postoperatively with regard to electrolyte changes, development of metabolic acidosis and urinary output.

Another problem following revascularisation of an acute ischaemic limb might be significant limb swelling. This may result in secondary muscle or nerve injury, venous compression, further oedema and compartment syndrome, leading to arterial compression and secondary ischaemia. To avoid this, the surgeon might prefer to perform a fasciotomy in conjunction with the embolectomy procedure [14]. Alternatively, the extremity can be assessed immediately and at regular intervals

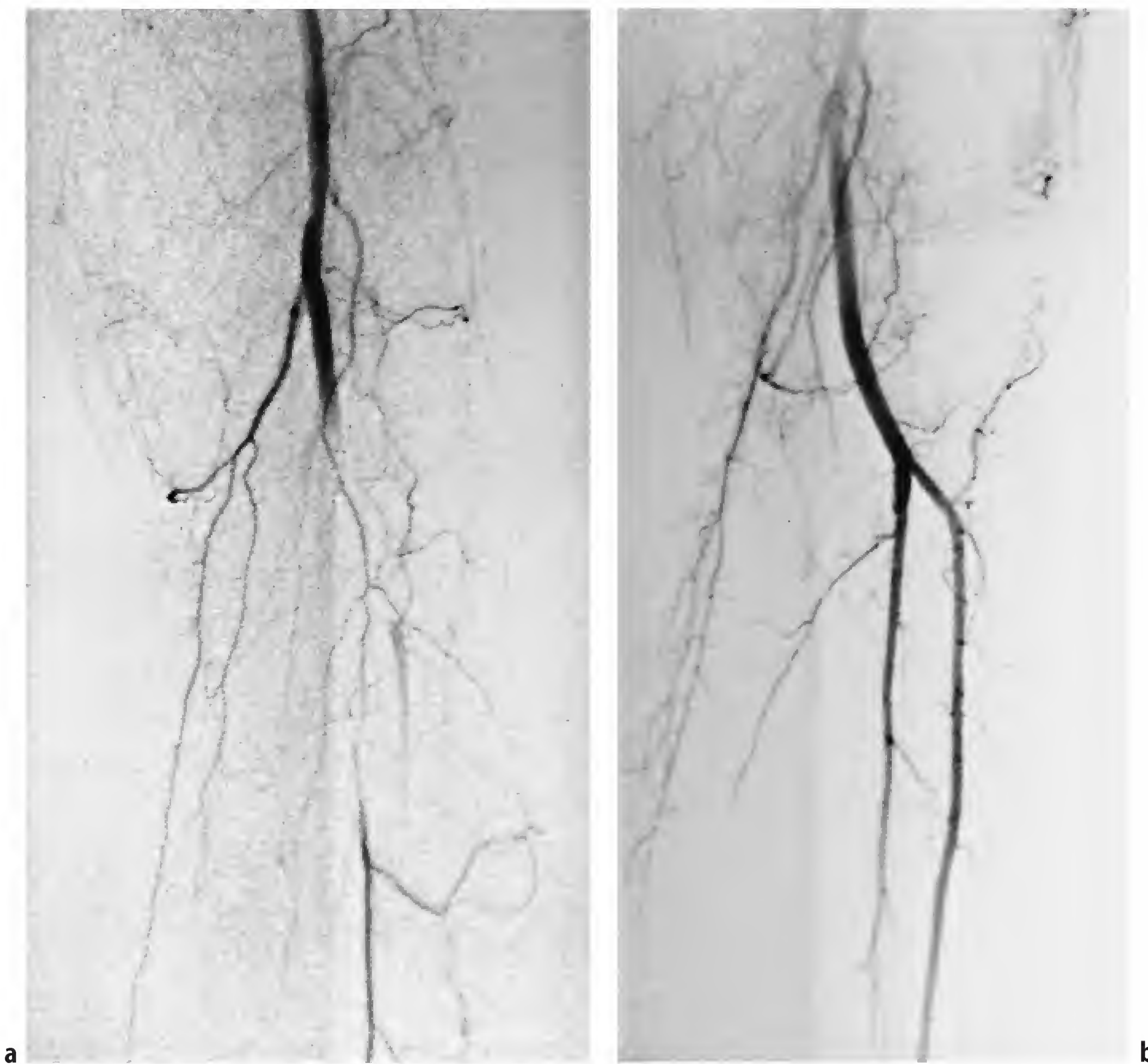


Fig. 11.1. **a** Embolic occlusion of the left popliteal artery; treatment consisted of percutaneous aspiration thromboembolectomy. **b** Normal patency of the popliteal, anterior tibial and peroneal arteries.

postoperatively for evolving compartment syndrome. As described in different textbooks, there are several ways of performing an adequate fasciotomy. The most important point here is that all four compartments should be decompressed.

Although concomitant fasciotomy can be preferable in some cases of prolonged acute ischaemia, the more conservative approach might avoid unnecessary fasciotomy and unaesthetic scars. Since a Fogarty catheter embolectomy can easily be carried out under local anaesthesia, this wait-and-see approach eliminates the need for systematic general anaesthesia, particularly for patients in a poor general condition [15].

Despite the fact that the value of postoperative compartmental pressure measurements has been documented by several teams [16, 17], the decision regarding subsequent fasciotomy is most frequently based upon individual preferences and prior clinical experience. **[Q5: D]**

Every effort should be made in the postoperative period to minimise the incidence of recurrent emboli. The patient should be treated with heparin or oral anticoagulants until the source of the embolus has been taken care of. **[Q6: B]** If

extensive investigation fails to show any correctable source, then long-term anticoagulation is indicated, except in the case of major contraindications.

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12. Blast Injury to the Lower Limb

Paul H. B. Blair, Adrian K. Neill and
Christopher T. Andrews

A 40-year-old male was admitted to the emergency room approximately one and a half hours after sustaining a blast injury to both lower limbs. He had been resuscitated at his local accident and emergency department prior to transfer. On arrival, his pulse was 120 bpm and his blood pressure 80/40 mm Hg.

Examination revealed that the patient had sustained significant blast injuries to both lower limbs with no obvious torso injuries. The left leg had sustained neurovascular damage above and below the knee with concomitant bone and soft tissue injury; there was no tissue perfusion below the knee. On the right side there was a large wound in the thigh extending anteriorly to the knee joint with profuse bleeding; bony fragments could be seen in the wound and the right foot was pale with no palpable pulses and slight reduction in sensation.

Question 1

The priorities for the care of this patient include:

A. Secure an airway, commence oxygen therapy and obtain adequate intravenous (IV) access.

B. Complete a full survey of the patient before transferring for further management.

C. Wait for blood result before deciding on transfer out of the emergency room.

D. Transfer the patient to theatre for definitive management during primary resuscitation.

E. Discuss treatment options with relatives.

Question 2

Which of the following are "hard" signs of vascular injury?

A. Limb pain.

B. Absence of pulses.

C. Pallor or cyanosis.

D. Cool to the touch.

E. Bruit or thrill.

Question 3

Which of the following statements relating to angiography are true?

A. Angiography should be performed in all patients to target surgery.

B. Angiography may be a useful tool in trauma patients with no hard signs of vascular injury.

C. Angiography is reserved for stable patients.

D. Angiography should only be performed in a radiology department.

E. The patient's pre-morbid condition should not influence the decision to perform angiography.

Question 4

For how long will the lower limb tolerate ischaemia?

A. 20–30 minutes.

B. 90–120 minutes.

C. 6–8 hours.

D. 16–20 hours.

E. 24–36 hours.

The patient was resuscitated as per ATLS (advanced trauma life support) protocol. Supplementary oxygen was administered in addition to obtaining additional IV access. Pressure dressings were applied to the open wounds and further assessment revealed an injury to the patient's right hand; no other significant injuries were present. The patient was transferred to the operating theatre.

Question 5

What are the primary aims of surgery in such a case?

A. To control life-threatening haemorrhage.

B. To prevent end-organ ischaemia.

C. To restore vascular continuity.

D. To preserve limb function.

E. To detect occult injuries.

Question 6

What factors will influence the decision to perform an amputation?

A. Patient's age.

B. Mechanism of injury.

C. Time to treatment.

D. Degree of contamination.

E. All of the above.

Question 7

Which of the following statements about complex vein repair are true?

A. Complex vein repair should never be undertaken in the trauma patient.

B. Complex vein repair should only be performed in the absence of major arterial injury.

C. Complex vein repair should be used to improve venous return in unstable patients.

D. Complex vein repair may prevent long-term limb dysfunction.

E. Intraluminal venous shunting is an acceptable intraoperative temporising measure.

In the operating theatre, under general anaesthesia, the patient was placed in the supine position. The lower abdomen and both legs were prepared and draped widely and intravenous broad spectrum antibiotics were administered. Closer examination revealed that the left leg had sustained extensive injuries. The foot and distal calf were cold, pale and mottled. There was a compound injury to the left femur and tibia with complete disruption of the superficial femoral artery, superficial femoral vein and extensive injury to the sciatic nerve. It was decided that primary amputation of the left limb was required. On examination of the right leg there was complete disruption of the distal superficial/popliteal artery, a ragged laceration of the popliteal vein and significant bruising to branches of the sciatic nerve. There was a shrapnel injury to the right hand involving the thumb and middle finger.

Immediate surgical steps were as follows: (i) a proximal thigh tourniquet was placed on the left leg to arrest haemorrhage prior to formal amputation. The laceration to the right lower leg was then extended distally to facilitate exposure of the neurovascular structures. Control of the superficial femoral and below-knee popliteal artery was obtained and a careful distal embolectomy performed. A Javid

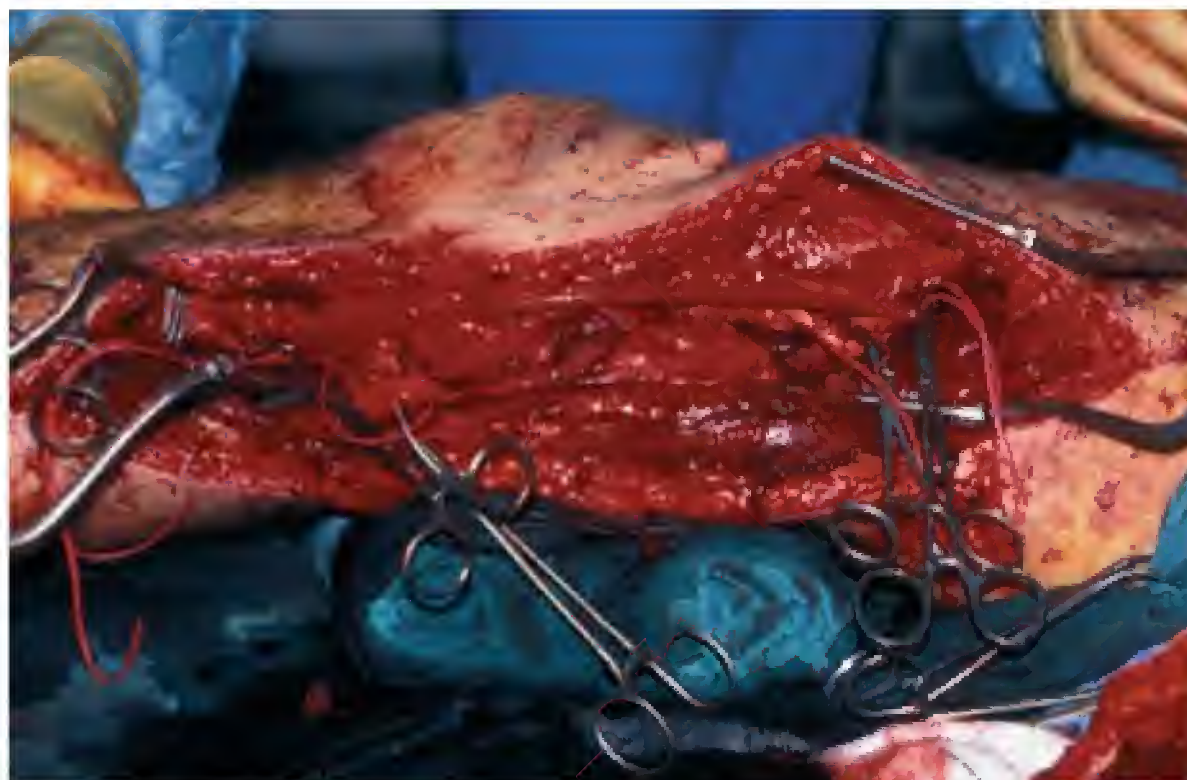


Fig. 12.1. Extended wound, medial aspect of right leg with a temporary intraluminal shunt between superficial femoral and below-knee popliteal arteries.

shunt was then placed between the right superficial femoral artery and right below-knee popliteal vessel (Fig. 12.1). Significant bleeding from a large defect in the popliteal vein occurred following shunt insertion; this was repaired using a lateral suture. The long saphenous vein was harvested from the left leg, prior to performing above-knee amputation. While the left above-knee amputation was being performed, the orthopaedic surgeons carefully assessed the right lower limb and placed a temporary fixation device traversing the right knee joint (Fig. 12.2). Having obtained bony stability, with an external fixator device, the temporary intraluminal shunt was removed and a definitive bypass performed using reversed left long saphenous vein graft. Formal fasciotomy was performed of the right lower leg using a standard lateral and medial approach; distal pulses were confirmed in the right foot. Further debridement of necrotic muscle was performed and the wound on the medial aspect was partially closed; the anterolateral wounds were debrided and irrigated, as were the fasciotomy sites, with sterile dressings being applied to both.



Fig. 12.2. A multidisciplinary approach. Bony stabilisation of right leg (after temporary intraluminal shunt placement) by the orthopaedic surgeons, simultaneous with left above-knee amputation by the vascular surgeons.

Question 8

In the absence of obvious haemorrhage, when is it appropriate to reinspect the wounds in the postoperative period?

A. 1–2 hours.

B. 4–6 hours.

C. 12–16 hours.

D. 24–48 hours.

E. 5+ days.

Postoperatively the patient was transferred to the intensive care unit where the right limb was elevated to reduce swelling. The right foot was left exposed to allow access for pedal pulses. Broad spectrum IV antibiotics were continued in addition to standard prophylaxis for deep vein thrombosis, and urine was checked for myoglobinuria. The patient was returned to the operating theatre within 48 hours for wound inspection and change of dressing. Eventually skin coverage of the right limb was obtained using a combination of split skin grafting and healing by delayed primary intention. Over the next few months the patient required complex orthopaedic surgery including the use of an Ilizarov frame device (Fig. 12.3). He was fitted with an above-knee prosthesis for his left leg and is now fully independent (Fig. 12.4).

Commentary

Lower limb injuries, due to penetrating trauma, can be devastating and occasionally may distract the clinician from less obvious but potentially life-threatening injuries to the head, neck and torso. It is important that some form of resuscitation protocol is followed such as the ATLS system to detect less obvious injuries. Time is of the



Fig. 12.3. Recovery. Healed traumatic and fasciotomy wounds after skin grafting; Ilizarov frame still in place.



Fig. 12.4. Rehabilitation. An excellent result for limb salvage (right leg) and learning to function with a prosthesis (left).

essence when managing vascular injuries. While delays rarely occur in patients with obvious haemorrhage, it is the prompt instigation of life-saving measures and ongoing diagnosis in parallel with transfer to the operating theatre for definitive care that reduces morbidity and mortality. **[Q1: A, D]**

The clinical manifestations of vascular injury have traditionally been divided into "hard" and "soft" signs (Table 12.1). **[Q2: B, E]**

Table 12.1. Signs of vascular injury. Updated 🕮

Hard signs	Soft signs
Absent pulse	Haematoma (small)
Bruit or thrill	History of haemorrhage at scene
Haematoma (large or expanding)	Peripheral nerve deficit
Distal ischaemia	

In general, preoperative arteriography may be used in the following situations: (1) to confirm the site and extent of vascular injury in stable patients whose clinical signs and symptoms are equivocal; and (2) to exclude vascular injury in patients with no hard signs, but who are considered to be at risk because of the proximity of the injury. The majority of patients with penetrating extremity trauma and the presence of a single hard sign should be transferred directly to the operating theatre. Possible exceptions to this rule include stable patients with multiple levels of injury, extensive bone or soft tissue injury, blast or shotgun injuries, potential injuries to the subclavian or axillary arteries and the pre-existence of peripheral vascular disease. Some centres report excellent results with emergency room angiography [1] while recent advances in endovascular technique facilitate high-quality imaging in the operating theatre. **[Q3: B, C]**

Inadequate tissue perfusion due to major vessel disruption is aggravated by hypovolaemic shock and associated bone and soft tissue injury. The resulting fall in tissue pO_2 increases capillary membrane permeability, with increased exudation of fluid into the interstitial space. Compromised muscle fibres swell within the fascial compartments, causing further resistance to blood flow, and swelling becomes traumatic when arterial repair and restoration of flow brings about reperfusion injury. The degree of reperfusion injury depends on the duration of ischaemia, and is mediated by the generation of free radicals, activation of neutrophils, and production of arachidonic acid metabolites. Eventually, the microvascular bed of the extremity may undergo widespread thrombosis [2]. It is generally accepted that a warm ischaemia time of more than 6–8 hours makes limb survival unlikely. **[Q4: C]** To achieve optimal results from emergency vascular repair, and to avoid complications such as compartment syndrome or contracture due to prolonged warm ischaemia and reperfusion injury, surgical exploration should be undertaken expeditiously.

A patient with complex lower limb injuries should be placed in a supine position on an operating table suitable for on-table angiography, if required, when clinical stability has been reached. Some form of warming device should be employed to maintain adequate body temperature. In lower limb trauma, both limbs should be prepared from umbilicus to toes; donor saphenous vein harvesting may be required from the contralateral limb, particularly if ipsilateral venous injury is suspected. Careful attention should be given to correct hypothermia, blood loss, electrolyte imbalance and coagulopathy.

The principal aims of emergency vascular surgery are to control life-threatening haemorrhage and prevent end-organ ischaemia. **[Q5: A, B]** An assistant should control haemorrhage using a pressure dressing until the patient is prepared and draped appropriately. Haemorrhage control can be difficult if the proximal vessels are not immediately apparent, and the use of a cephalad incision through virgin territory may be a reasonable alternative to obtain rapid proximal control. Care should be taken when making additional incisions, particularly if it seems likely that plastic surgery will be required at a later date. When access to the proximal or distal vessel is difficult, temporary control can be gained by careful cannulation and inflation of an embolectomy catheter. It is important that the surgeon cooperates fully with the anaesthetist during surgery as it may be necessary to pack the wound for a few minutes to facilitate intravenous fluid resuscitation before proximal vascular control can be obtained. Complex lengthy operations should be avoided in unstable patients and damage limitation surgery should be considered in patients with significant metabolic acidosis, coagulopathy and/or hypothermia.

The use of a temporary intraluminal vascular shunt should be considered in the majority of limb vascular injuries and is particularly important in complex cases with associated bone and soft tissue injury.

Temporary shunts for arterial and venous injuries have been employed in Belfast since the late 1970s [2]. A considerable body of evidence continues to support the use of these intravascular shunts in the management of both penetrating and blunt major vascular trauma [3–6]. Before securing the shunt between the proximal and distal arteries, a careful embolectomy should be performed to remove any thrombus in the distal vessel. If a venous injury is encountered, then an additional shunt can be employed to facilitate venous return. In the absence of coagulopathy or ongoing haemorrhage we use intravenous heparin routinely. Recent evidence has shown clearly that delayed renewal of venous flow in combined arterial and venous injury compounds ischaemia–reperfusion injury and causes remote lung injury [7]. The advantages of shunting artery and vein are the early restoration of blood flow and venous return, respectively, thus avoiding the complications of prolonged ischaemia and ischaemia–reperfusion injury while ensuring that an optimal vascular repair can be performed.

In patients with concomitant fractures, accurate internal or external fixation of the fracture can be performed with the shunt secured carefully with sloops before definitive vascular repair is performed. This avoids the dilemma of unnecessary haste for both the orthopaedic and vascular surgeons, ensures that a vein graft will be of optimal length, and eliminates the risk of graft disruption during fracture manipulation. Autologous vein is our preferred bypass conduit in the majority of cases because of its durability and suitability in a potentially contaminated wound. Satisfactory results, however, have been reported using synthetic grafts and in critically ill, unstable patients this may be a preferable option [8].

The acute management of high energy limb trauma can be challenging and significant morbidity and mortality can occur following failed attempts at limb salvage. A number of scoring systems have been devised in an attempt to assist the clinician's decision to either amputate or perform a limb-salvage procedure [9–13]. In each of the systems, a score is assigned based on a range of differing criteria including patient age, "mechanism of injury", time to treatment, degree of shock, warm ischaemia time and the presence of local injuries to the following structures: major artery, major vein, bone, muscle, nerve, skin, and degree of contamination. **[Q6: E]** All of these scoring systems demonstrate a much higher degree of specificity than sensitivity and are more useful in highlighting the patients who should be considered for a limb-salvage procedure, than identifying those who should proceed straight to primary amputation. Indeed a number of studies have challenged their use at all [14, 15].

It is the authors' opinion that scoring systems can help the surgeon perform a detailed assessment of a complex limb injury. However, the decision to perform a primary amputation must be judged individually in each case. Extensive nerve injuries have a particularly poor prognosis and it is important that such injuries, where possible, are documented before taking the patient to the operating theatre. The patient's life should never be put at risk in a futile attempt to save a severely compromised limb. Where possible, additional specialties such as orthopaedics and plastic surgery should be involved in the decision to perform a primary limb amputation, particularly in a case of upper limb trauma.

Venous injuries can be difficult to manage. Prior to World War II, the traditional treatment for lower extremity venous injuries was ligation. This custom was challenged by Debakey & Simeone [16] in 1946 with an analysis of WWII battle injuries.

Since then a number of clinical and laboratory investigations have confirmed that ligation of major veins in conjunction with repair of a traumatically injured arterial system leads to significantly poorer clinical outcomes, such as decreased function or even limb loss [17, 18]. Where possible vein repair should be attempted, particularly in the presence of significant lower limb arterial injury, in an attempt to reduce venous hypertension and associated morbidity. While there are few data regarding the long-term outcome of venous repairs, it is the authors' impression that maintaining venous patency, in the initial few days after injury, can significantly help reduce acute post-injury swelling. If the superficial femoral vein requires ligation, it is important to maintain patency of the ipsilateral long saphenous and profunda femoris veins. Complex vein repair should never be attempted in unstable patients who have sustained major blood loss and have significant problems with hypothermia and coagulopathy. In more stable patients, however, temporary intraluminal venous shunting can facilitate the construction of larger calibre panel grafts obtained from the contralateral long saphenous vein. **[Q7: D, E]**

Postoperative management of patients with complex limb injuries is critically important. The majority of these patients have been transferred immediately to the operating theatre and it is important that a thorough search for occult injuries is performed on admission to the intensive care unit. These patients are at risk of developing multiple organ dysfunction syndrome as a result of their large transfusion requirements and likely reperfusion injury sustained [19, 20]. It is important that the vascular surgeon communicates clearly with the staff in the intensive care unit regarding the presence or absence of distal pulses, to ensure that vascular repair remains patent. Young trauma patients with normal blood pressure and temperature should have a palpable distal pulse. If there is any doubt regarding the integrity of the vascular repair, the dressings should be removed and a careful assessment performed by a vascular surgeon using handheld Doppler and/or portable ultrasound device.

Wounds should be reinspected 24–48 hours after initial surgery and at that stage definitive plastic surgery may be required to obtain soft tissue and skin cover. **[Q8: D]** Some centres advocate a selective policy with regard to fasciotomy based on compartmental pressures, while many continue to advocate a more liberal policy based on clinical grounds. Prolonged ischaemia time, combined arteriovenous injuries, complex injuries including bone and soft tissue destruction and crush injuries remain absolute indications for fasciotomy. The avoidance of compartment syndrome and restoration of limb function far outweigh the low morbidity associated with liberal use of fasciotomy. These patients are at significant risk of wound and other nosocomial infections and prolonged antibiotic use may be required.

The management of patients with complex injuries can be difficult; however, timely surgery and the involvement of a multidisciplinary team can produce rewarding results. One possible criticism of the above care could be failure to use the great toe, from the amputated left lower limb, to replace the patient's right thumb.

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13. Endoluminal Treatment of Traumatic Arteriovenous Fistula of the Axillary Artery

Jonathan D. Woody and Rodney A. White

A 21-year-old male sustained a gunshot wound to the anterior right chest. The entrance wound was at the midclavicular line. He was hemodynamically stable upon arrival at the emergency department. He underwent initial resuscitation with intravenous fluids. There was no evidence of hemothorax or pneumothorax on chest X-ray. The right radial pulse was present but diminished when compared with the left. The patient had minor neurological symptoms in the right upper extremity, which consisted mainly of weakness of the interosseous muscles of the hand. An arteriogram was obtained, which revealed an arteriovenous fistula (AVF) of the right axillary artery and vein (Fig. 13.1a). The patient remained hemodynamically stable throughout the evaluation period.

Question 1

Acquired AVF is most commonly the result of:

A. Penetrating trauma.

B. Percutaneous puncture.

C. Erosion of arterial aneurysm.

D. Periarterial abscess.

E. Neoplasm.

Question 2

Which of the following are possible complications of AVF?

A. Bacterial endarteritis at the site of AVF.

B. Peripheral arterial insufficiency.

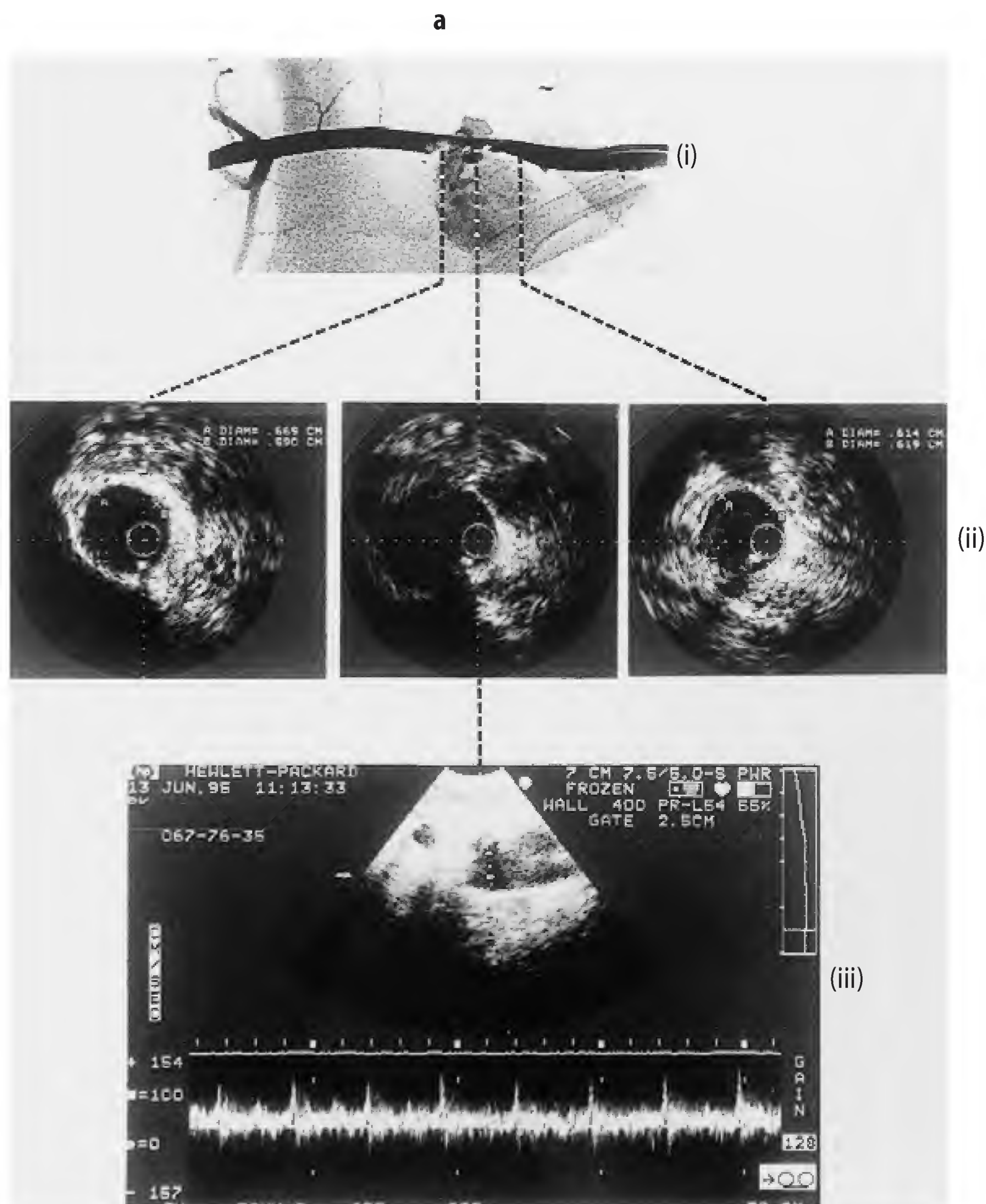


Fig. 13.1. a Pre-treatment and **b** post-treatment images of an AVF of the right axillary artery after a gunshot wound. **i** Arteriography, **ii** intravascular ultrasound, and **iii** duplex grey-scale ultrasound demonstrate the AVF before treatment and the complete exclusion of the fistula after treatment. (Reprinted from J Vasc Surg, vol. 24, White RA, Donayre CE, Walot I, et al., Preliminary clinical outcome and imaging criterion for endovascular prosthesis development in high-risk patients who have aortoiliac and traumatic arterial lesions, pages 569–571, © 1996, with permission from The Society for Vascular Surgery.) 📖

C. Venous congestion, venous valvular insufficiency, venous stasis, venous varicosities and edema formation.

D. Tachycardia.

E. Cardiomegaly.

F. Congestive heart failure.

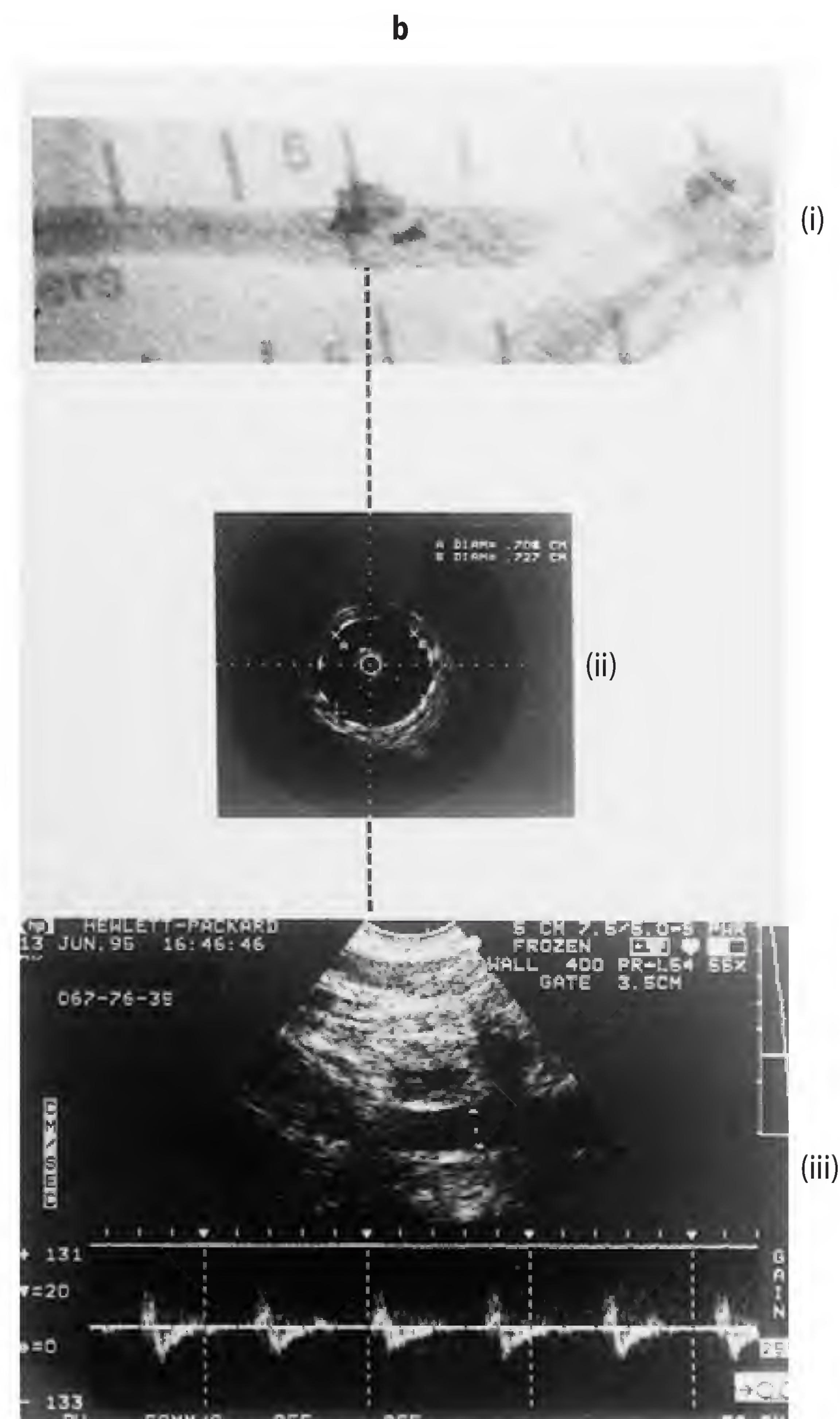


Fig. 13.1. *(continued)*

Question 3

What is the gold standard for the diagnosis of AVF?

A. Magnetic resonance imaging (MRI)/magnetic resonance angiography (MRA).

B. Computed tomography (CT).

C. Color-flow duplex ultrasound.

D. Arteriography.

Treatment

The patient was transferred to the endovascular operating suite and placed under general anesthesia. He was positioned on the interventional table with his right arm on an arm board. A surface ultrasound was performed, which confirmed the location of the AVF. The right arm and chest were prepared into a sterile field, and the abdomen and upper legs were prepared into a separate sterile field. The right brachial artery and vein were surgically exposed and isolated. A sheath was inserted percutaneously into the right femoral artery. Under fluoroscopic guidance, a guidewire was passed through the femoral sheath into the right axillary artery. It was then passed across the injured segment of the right axillary artery and into the right brachial artery to the surgical site. The wire was removed through a small transverse arteriotomy, and a hemostatic sheath was inserted into the artery over the wire.

The length of the axillary artery was interrogated with intravascular ultrasound. The diameter of the axillary artery was 7 mm. A 4-cm segment of deep brachial vein was obtained and sutured to an appropriately sized Palmaz stent. This was crimped onto an expandable balloon and, under fluoroscopic guidance, passed over the guidewire to the site of injury. The balloon was expanded, and the endoluminal device was deployed. The balloon was deflated and removed. The axillary artery was again interrogated with intravascular ultrasound, which demonstrated excellent apposition between the stent and the arterial wall. Complete exclusion of the fistula was documented with both intravascular ultrasound and arteriography (Fig. 13.1b). Flow was then restored to the arm. The arm wound was closed surgically, and pressure was applied to the femoral puncture site. Before awakening the patient, surface ultrasound confirmed the closure of the fistula and normal flow through the axillary artery.

Question 4

Which of the following is not considered to be one of the core principles of treatment of AVF?

A. Complete closure of the fistula.

B. Restoration of normal arterial flow.

C. Ligation of the arterial inflow

D. Restoration of normal venous flow.

Commentary

AVF is an abnormal communication between an artery and vein. By far the most common cause of acquired AVF is trauma. This is usually the result of a penetrating injury secondary to a knife or bullet, but it can also result from blunt trauma. Iatrogenic injury from percutaneous puncture can also cause AVF. In addition, AVF can be caused by erosion of an arterial aneurysm into an adjacent vein, by a periarterial abscess or by a neoplasm, although these are rare. **[Q1: A]**

Potential complications of AVF can be considered on both a peripheral and a systemic level. Peripheral effects may include peripheral arterial insufficiency, dilated and thickened proximal arterial wall (predisposing to infection, i.e. bacterial endarteritis), venous congestion, venous valvular insufficiency, venous stasis, varicosities, edema, and skin and soft tissue changes. Systemic effects may include increased cardiac output, tachycardia, cardiomegaly, congestive heart failure, increased pulse pressure, decreased diastolic pressure, increased blood volume, increased central venous pressure, and increased stroke volume. **[Q2: A, B, C, D, E, F]**

The diagnosis of AVF is relatively straightforward. An audible bruit and palpable thrill are often found, especially in peripheral lesions. The gold standard for diagnosis is arteriography. The most important point is to delineate clearly the anatomy of the fistula so that treatment may be planned. Other diagnostic modalities include color-flow duplex ultrasound, CT, and MRI and/or MRA. **[Q3: D]**

Treatment of AVF is best carried out early rather than late. The longer an AVF is present, the more likely the patient will be to develop complications. Further, the longer it exists, the more likely it is to develop significant collateral flow, which increases the complexity of the repair. The principles of repair, whether conventional or endovascular, are the same: complete closure of the fistula and restoration of normal arterial and venous flow. **[Q4: C]** Conventional repair can be undertaken in a number of ways but will not be described here since conventional repair is not the focus of this chapter.

The first report of successful endovascular repair of an arterial injury was by Becker et al. [1]. Further reports by Parodi and Barone [2], Marin et al. [3] and May et al. [4] have demonstrated a high rate of technical success with persistent fistula closure, excellent long-term patency, and a low incidence of complications. Endovascular treatment of AVF is ideal because most of these injuries are localized to a small segment of artery with normal vessel wall on each end. Endovascular treatment of these lesions also avoids the major morbidity of conventional open repair, which can be significant. Endovascular access is achieved at a site remote from the area of injury and can be performed percutaneously in many cases. There is little damage to surrounding structures since only the affected vessel is manipulated. If endovascular treatment is successful, then the benefits for the patient include a shorter hospitalization period and a quicker return to normal activity. The only major concern is long-term patency and function of the endoluminal stent graft, since these studies are not yet available. However, due to the relatively short length of the stent graft, its placement in large-diameter vessels, and the high flow rates in these vessels, it is reasonable to assume that the long-term patency would be good [5]. It is of note that our patient was hemodynamically stable. Although endovascular technology continues to evolve and improve, it must be emphasized that a hemodynamically unstable patient who has sustained penetrating trauma should undergo immediate open surgical exploration.

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14. Cardiovascular Risk Factors and Peripheral Arterial Disease

Stella S. Daskalopoulou and Dimitri P. Mikhailidis

A 62-year-old man with intermittent claudication was referred for vascular risk factor modification. He had no history of myocardial infarction (MI) or stroke. He was smoking 20 cigarettes/day. His family history was negative for premature vascular events. He was not taking any medication. He was advised to start aspirin 75 mg/day, but he stopped taking these tablets because of "stomach discomfort". The patient's total cholesterol was 228 mg/dl (5.9 mmol/l).

His blood pressure required treatment with amlodipine and a thiazide diuretic. The patient eventually stopped smoking after referral to the smoking cessation clinic in our hospital.

Question 1

Which of the following investigations would you order?

A. Fasting serum glucose.

B. Urine glucose to make a diagnosis of diabetes mellitus.

C. Fasting serum triglycerides.

D. Fasting serum high-density lipoprotein cholesterol (HDL-C).

E. Thyroid function tests.

A. Requesting a fasting serum glucose level is an essential test in all patients with vascular disease. In this case the fasting glucose was 87 mg/dl (4.8 mmol/l); this is satisfactory.

Interpretation of fasting glucose values:
There are three categories in which a patient can be placed relative to fasting serum glucose levels:

- *Normal:* fasting glucose =110 mg/dl (=6.0 mmol/l).

Table 14.1. Features of metabolic syndrome*. Updated

1. *Abdominal obesity (waist circumference):*
Men >102 cm
Women >88 cm
2. *Triglycerides*
=150 mg/dl (=1.7 mmol/l)
3. *High-density lipoprotein cholesterol (HDL-C):*
Men <40 mg/dl (<1.0 mmol/l)
Women <50 mg/dl (<1.3 mmol/l)
4. *Blood pressure:* <130/<85 mm Hg
5. *Fasting glucose:* =110 mg/dl (=6.0 mmol/l)

*According to the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III guidelines [1], any 3 or more of these 5 features are diagnostic of the metabolic syndrome. Other factors that may coexist in these patients include a family history of type 2 diabetes, South Asian ethnicity, decreased physical activity, smoking, elevated serum urate levels and evidence of fatty liver (abnormal levels of aminotransferases, ALT/AST).

There is evidence (post-hoc analysis) from the Scandinavian Simvastatin Survival Study (4S) that IFG and diabetic patients benefit from treatment with simvastatin [2]. More recently, a trial in type 2 diabetic patients without established vascular disease showed a beneficial effect of atorvastatin 10 mg/day (vs. placebo) in reducing the risk of first cardiovascular events, including stroke [3].

Both diabetes and metabolic syndrome are common in patients with peripheral arterial disease (PAD) [4]. Furthermore, both diabetes and PAD are considered as coronary heart disease (CHD) equivalent and need to be treated aggressively [1].

- *Impaired fasting glucose (IFG):* fasting glucose 110–125 mg/dl (6.0–6.9 mmol/l).
- *Diabetes mellitus:* fasting glucose =126 mg/dl (=7.0 mmol/l).

IFG is associated with an increased risk of vascular events and conversion to diabetes mellitus. Furthermore, a glucose level in the IFG range can be one of the features of the metabolic syndrome (also known as insulin resistance or Reaven's syndrome) [1] (Table 14.1).

B. This patient's urine was tested when he was first seen in outpatients. The renal threshold for glucose is a serum level of about 180–200 mg/dl (10–11 mmol/l). Therefore, testing urine for glucose will not detect IFG or early/mild diabetes. Clinicians must not rely on a urine glucose test to exclude IFG or early diabetes. In view of the serum glucose value (see **A**, above), it is not surprising that the urine glucose test was negative. However, testing the urine was an opportunity to exclude proteinuria, another indicator of vascular risk.

C. The fasting triglyceride level in this patient was 141 mg/dl (1.6 mmol/l) – this is satisfactory.

Interpretation of fasting triglyceride values:
There has been considerable confusion regarding the importance of triglycerides. There are several reasons for this, including:

- *Interactions with other lipid variables:* serum triglyceride and HDL-C levels are inversely related. HDL-C is a "protective" lipoprotein.
- *Interactions with potential risk factors:* elevated serum triglyceride levels are associated with impaired fibrinolysis and possibly elevated plasma levels of fibrinogen. Both type 2 diabetes and metabolic syndrome are associated with raised serum triglyceride levels.

Table 14.2. Secondary causes of hypertriglyceridaemia/hypercholesterolaemia. Updated 📖

Excessive alcohol intake
Diabetes mellitus
Hypothyroidism
Some types of liver disease
Some types of renal disease
Obesity/diet
Drugs: beta-blockers, thiazides, oestrogens, anabolic steroids, corticosteroids, tamoxifen, protease inhibitors, retinoids, ciclosporin

- *Triglyceride levels vary considerably within any one individual:* this variability includes the fact that fasting triglycerides may be considerably lower than non-fasting levels in some patients. There is evidence that postprandial triglyceride levels also predict vascular risk, but this measurement is not easily standardised. Therefore, assessment of triglyceride status is best represented by a fasting sample (14-h overnight fast; water only allowed).

The current opinion is that fasting serum triglyceride levels are independent vascular risk factors [5]. Hypertriglyceridaemia is often associated with secondary causes that aggravate the patient's tendency to this type of dyslipidaemia (Table 14.2). These causes need to be addressed.

Fasting triglyceride levels are defined in the NCEP ATP III guidelines [1]:

- *Borderline high:* 150–199 mg/dl (1.7–2.2 mmol/l).
- *Moderately elevated:* 200–499 mg/dl (2.3–5.6 mmol/l).
- *Severe hypertriglyceridaemia:* =500 mg/dl (=5.6 mmol/l).

According to these guidelines [1], the treatment priority for cases with severe hypertriglyceridaemia shifts from LDL-C to the triglyceride levels. This is because of the increased risk of acute pancreatitis associated with severe hypertriglyceridaemia [1]. For milder hypertriglyceridaemia, the priority for treatment remains the LDL-C level [1].

D. The fasting HDL-C level in this patient was 46 mg/dl (1.2 mmol/l) – this is satisfactory.

Interpretation of fasting HDL-C values:

A raised HDL-C level is a protective factor, whatever the levels of other lipid variables [1, 6, 7]. The recent NCEP ATP III guidelines [1] recommend that HDL-C levels should ideally be =40 mg/dl (=1.0 mmol/l) [1, 6, 7]. A low HDL-C level is also predictive of the risk of stroke [8, 9]. The importance of HDL-C in reducing the risk of vascular events is supported by the findings of a secondary prevention trial (VA-HIT) [9].

E. The thyroid function tests were normal.

It is useful to routinely assess thyroid function in dyslipidaemic patients. This is because hypothyroidism is not uncommon and it is associated with dyslipidaemia (see Table 14.2). There is also some evidence showing that hypothyroid patients are

Table 14.3. CHD equivalents according to the NCEP ATP III guidelines [1]. Updated

Peripheral arterial disease
Abdominal aortic aneurysm
Symptomatic carotid artery disease
Diabetes mellitus
Multiple risk factors conferring a calculated risk for a vascular event >20 per cent over the next 10 years

more likely to have "muscle-related" side effects if they are given a statin. Hypothyroidism can also be difficult to spot unless the clinical features are obvious. Replacement with thyroxine is usually associated with a beneficial change in the lipid profile and body weight.

Question 2

What drug would you use to treat this patient's dyslipidaemia? What are your target levels?

The main target for lipid-lowering treatment is the LDL-C level. Since PAD is considered a coronary heart disease (CHD) equivalent [1] (Table 14.3), the LDL-C target is 100 mg/dl (2.6 mmol/l) in the USA [1] and 96 mg/dl (2.5 mmol/l) in Europe [10]. The NCEP ATP III guidelines were revised in 2004 to include an optional LDL-C target of 70 mg/dl (1.8 mmol/l) for very high-risk patients [11].

As explained above, the HDL-C and triglyceride levels are secondary targets.

A full fasting lipid profile should be obtained before making any decision regarding treatment. In the case presented above, the fasting values were: total cholesterol = 228 mg/dl (5.9 mmol/l), HDL-C = 46 mg/dl (1.2 mmol/l), LDL-C = 155 mg/dl (4.0 mmol/l) and triglycerides = 141 mg/dl (1.6 mmol/l).

The drug of choice is a statin to achieve the LDL-C target. Statins also improve HDL-C and triglyceride levels, although these latter effects may be small.

Three statins have extensive trial-based evidence in terms of reduced mortality: atorvastatin, pravastatin and simvastatin. There is also evidence that treatment with statins decreases morbidity and mortality and improves symptoms in patients with PAD [4, 12].

There is convincing evidence that statins reduce the risk of stroke [12–14]. Several studies have also shown that aggressive lipid lowering is associated with a reduced progression of atherosclerotic carotid artery disease [13, 14]. Patients with PAD are likely to have some degree of carotid artery disease. PAD is also a strong predictor of the risk of stroke.

Question 3

What modifiable risk factors would you like to address in a high-risk patient, as in this case?

Smoking

Smoking cessation is of paramount importance. The vast majority of PAD patients are, or have been, smokers. Furthermore, smoking is associated with adverse effects on several variables that predict vascular events. For example, smoking can lower serum HDL-C levels, raise serum triglyceride levels, increase insulin resistance and elevate plasma fibrinogen concentrations [15]. Plasma fibrinogen is an independent predictor of the risk of MI and stroke. Smoking may even predict the progression of PAD and graft occlusion after infrainguinal bypass surgery [16]. There is evidence that the vascular risk is greater in smokers than in non-smokers, despite the use of statins [17].

There is a need to establish smoking cessation clinics where specialist care can be delivered. All clinicians should try to motivate patients to quit by spending a few minutes explaining why smoking is harmful to them. In PAD, quitting may improve claudication and reduce the risk of vascular events.

Antiplatelet Agents

This patient could not tolerate aspirin. It is estimated that this problem arises in 10–15 per cent of patients who are prescribed aspirin. There are several alternatives:

- "Cover" aspirin with a proton pump inhibitor (e.g. omeprazole).
- Eradicate *Helicobacter pylori* infection, if present.
- Use clopidogrel: the effectiveness of clopidogrel is based on the findings of major trials (e.g. CAPRIE, CREDO and CURE), but there is no study specifically designed to assess the effectiveness of this drug in PAD [18]. However, patients with PAD had significantly fewer events on clopidogrel than on aspirin in the CAPRIE trial. Unfortunately, this conclusion is limited by the fact that patient subgroup analysis was not included in the original trial protocol [18].

Due to his intolerance of aspirin, this patient was prescribed clopidogrel 75 mg/day. He tolerated this antiplatelet agent without any problems.

Blood Pressure

Strict control of blood pressure in high risk patients is essential [19]. In order to achieve this objective, there may be a need to use several antihypertensive drugs. Some general recommendations are appropriate:

- Several experts suggest that angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers should be avoided or used with caution in PAD because these patients may have renal artery stenosis. If an ACE inhibitor or angiotensin II receptor blocker is used, the plasma creatinine concentration should be monitored soon after starting treatment.
- There is still some debate as to whether beta-blockers adversely affect the circulation in the lower limbs of patients with PAD. It would appear reasonable, however, to use a beta-blocker in post-MI patients with PAD.

- Some blood pressure drugs exert beneficial or adverse effects on lipid levels, haemostatic factors and perhaps more importantly, the long-term risk of developing diabetes.

Glucose Status

This topic was discussed above. It is also important to note that if the patient is diabetic, the blood pressure targets become stricter, especially if proteinuria is present.

Lipids

This topic has been discussed above.

Emerging Risk Factors

These factors [1, 4] include:

- *Lipoprotein (a) (Lp(a)):* there is evidence that Lp(a) is a marker of vascular risk, especially in patients with a raised serum LDL-C. Raised Lp(a) levels may also predict the risk of restenosis after surgery for PAD [16]. Serum Lp(a) levels are difficult to lower, but the risk associated with this abnormality may decrease if the LDL-C level is markedly reduced. Correcting hypothyroidism is associated with a fall in serum Lp(a) levels. Similarly, postmenopausal hormone replacement therapy (HRT) may reduce serum Lp(a) concentrations. There are, as yet, no intervention trials to show that lowering serum Lp(a) levels (e.g. by using high doses of nicotinic acid) is associated with fewer vascular events.
- *Homocysteine:* raised plasma levels of homocysteine are thought to predict vascular risk possibly by acting synergistically with established risk factors. The link between homocysteine and PAD appears to be stronger than with CHD [4]. There are, as yet, no intervention trials to show that lowering plasma homocysteine levels (e.g. by folic acid, vitamin B12 or B6 supplements) is associated with a reduced risk of vascular events.
- *Haemostatic and fibrinolytic factors:* there is strong evidence showing that the plasma fibrinogen concentration is an independent predictor of vascular risk. The levels of this coagulation factor also predict the progression of PAD and possibly the risk of restenosis following bypass surgery [16]. Plasma fibrinogen levels can be lowered by some fibrates used to treat dyslipidaemia. However, as with other emerging risk factors, no trial-based evidence is available to show that lowering fibrinogen levels is associated with a decreased risk of vascular events. There is less evidence linking fibrinolytic factors with vascular risk.
- *Markers of inflammation (e.g. C-reactive protein, CRP):* serum CRP levels predict the risk of a vascular event even when there is no vascular disease present or when lipid levels are "normal". We do not know whether CRP just reflects the inflammatory component of atherosclerosis or whether it is actually involved in its pathogenesis. Statins and fibrates lower serum levels of CRP [20]. Recent evidence suggests that we should also consider CRP levels (in the high sensitivity range) as a target for treatment [21].

Question 4

Is it relevant to monitor renal function in this patient?

Yes, because about 33 per cent of PAD patients have atherosclerotic renal artery stenosis [4]. It is therefore important to consider the presence of this condition, especially if the renal function tests are abnormal. There is evidence that renal and vascular disease progress in parallel [22]. Increased plasma creatinine levels are associated with a higher risk of vascular events, even if these values are in the upper end of the reference range. There is evidence that statins exert a renoprotective action in patients with CHD or PAD [23, 24]. Impaired renal function may contribute to hyperuricaemia and hyperhomocysteinaemia [25]. These variables may predict increased vascular risk.

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15. Angioplasty for Critical Arterial Stenosis

Lars Norgren

A 62-year-old man, formerly a shopkeeper and a heavy smoker (26 cigarettes daily until 5 years ago when he succeeded in giving up), presented with a history of leg pain. Since retirement 2 years ago, he had started going for walks in the nearby countryside. From the start, he could walk for 5 miles, but he felt some pain in his left calf, which reduced after slowing down.

Question 1

Which of the following statements are correct?

A. This symptom is typical for both intermittent claudication and knee joint arthrosis.

B. This man presents exceptional symptoms: in the vast majority of cases, both legs are affected.

C. Intermittent claudication affects about 5 per cent of the male population over the age of 55 years.

D. This man should be advised to stop walking to reduce the risk of pain.

E. The symptom is consistent with an occlusion or a critical stenosis of the superficial femoral artery (SFA).

The man did not seek advice at that stage, but 2 years later, he suddenly experienced more severe pain in the left calf when he woke up in the morning. This happened 3 days before presentation. The pain disappeared slowly, but walking was then restricted to about 20 m, at which point the pain started. He felt some numbness in the toes, and on awaking in the morning there was some pain in the foot, which disappeared when he stood up.

Question 2

What is the most likely reason for the new symptom?

A. Muscle rupture.

B. Deep vein thrombosis.

C. Thrombosis of an artery or collateral artery.

D. Minor stroke.

E. Aggravation of knee joint arthrosis.

At examination, femoral pulses were present but no popliteal or foot pulses could be felt. Doppler flow was detected in the left posterior tibial artery, and in the right posterior tibial artery and dorsalis pedis artery (DPA). Ankle brachial pressure index (ABPI) of the affected leg was 0.4; that of the right leg was 0.8.

An angiography was performed, depicting lesions as shown in Fig. 15.1, as less than 50 per cent stenosis of the left external iliac artery (EIA), two short stenoses of more than 70 per cent of the left SFA, and an occlusion of a deep femoral artery branch. On the right side, there was one stenosis of the SFA of about 70 per cent. There were also short occlusions of the anterior tibial artery bilaterally.

Immediately following the diagnostic angiography, it was decided to perform a balloon percutaneous transluminal angioplasty (PTA) of the stenoses of the left SFA. The right leg was left untreated.

Question 3

Which of the following statements are correct?

A. There is normally no indication to treat an asymptomatic limb.

B. It is never possible to reopen occlusions of the deep femoral artery branches.

C. A stenosis of less than 50 per cent is not critical and does not normally need to be treated.

D. The critical stenosis of the SFA in our case is probably the primary reason for this patient's initial symptoms.

E. The deep femoral artery occlusion could well be the reason for the aggravation of the patient's symptoms.

At follow-up after 30 days, the patient had improved and could walk his daily 5 miles, albeit with some tolerable claudication.

Question 4

Which investigations are relevant for the follow-up of this case?

A. Digital subtraction angiography.

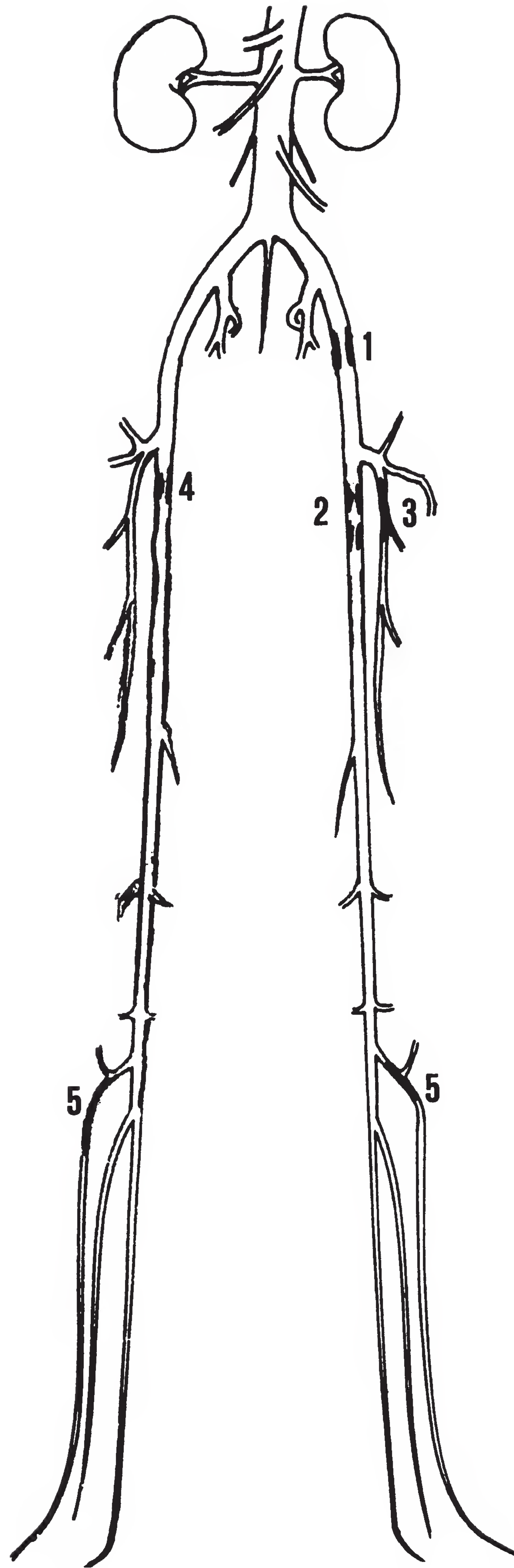


Fig. 15.1. Lesions found on angiography. 1, <50 per cent stenosis of left external iliac artery; 2, two short stenoses (<70 per cent) of left superficial femoral artery; 3, occlusion of a left deep femoral artery branch; 4, short stenosis (70 per cent) of the right superficial femoral artery; 5, short occlusions of anterior tibial artery bilaterally. 📖

B. ABPI measurement.

C. Spiral CT angiography.

D. Duplex investigation.

E. Laser Doppler flux measurement.

Question 5

What advice and adjunctive treatment should be given to this patient?

A. Continuing exercise, but stopping if pain appears.

B. Antiplatelet drug treatment.

C. Antiplatelet drug + warfarin treatment.

D. Avoid cycling. Hip flexion increases the risk of new femoral stenotic or occlusive lesions.

E. Continuing exercise.

Commentary

Intermittent claudication affects about 5 per cent of the male population over 55 years and about 2.5 per cent of the female population [1]. Calf pain during walking, caused by occlusion or stenosis of the SFA, is the most common symptom; in the majority of cases, only one leg is symptomatic to start with. **[Q1: C, E]** Only about 25 per cent of patients suffering intermittent claudication deteriorate to critical limb ischaemia (CLI) [2, 3]; the majority remain stable or even improve. The major risk is cardiovascular complications.

When a sudden deterioration appears, as in our case, a thrombotic event is most likely. **[Q2: C]**

An ABPI of 0.8 indicates the presence of asymptomatic peripheral arterial occlusive disease (PAOD). Symptoms are hidden due to the walking restriction from the more severely diseased contralateral leg. An ABPI of 0.4 can be consistent with CLI, but symptoms are not convincing. Usually, the definition of chronic CLI requires stable symptoms for at least 2 weeks [4]. The symptoms fit more appropriately into the definition of acute ischaemia, with a viable, unthreatened leg [5].

Critical stenosis produces a measurable pressure gradient and/or reduced blood flow. Usually, more than a 75 per cent reduction of the cross-sectional area is required, which means more than a 50 per cent reduction of the luminal diameter. **[Q3: A, C, D, E]** Also, a less pronounced grade of stenosis may produce haemodynamic disturbances and symptoms, especially during exercise. Two or more stenoses after each other may also become significant at a lesser grade of stenosis.

In principle, non-disabling claudication symptoms do not justify interventional treatment. Had this patient sought advice before the acute symptoms appeared, he would have been told to continue exercising and to try to continue walking when pain appeared. The acute symptoms changed the scene, although the leg was not in danger. In this situation, an angiography should be performed soon to enable treatment. A duplex examination could be carried out first as a screening procedure.

PTA was performed on the two subsequent and critical SFA stenoses, while the uncritical CIA stenosis was left. If in doubt about whether to treat a stenosis, than a measurement of the pressure gradient over such a stenosis is valuable. If this gradient is less than 10 mm Hg (mean pressure), then there is no indication to dilate.

The occluded profunda branch is a delicate problem. It might be a thrombotic occlusion, preventing collateral blood flow to the distal SFA. One could consider

leaving a catheter in the thrombus and starting thrombolysis; however, this decision is at the surgeon's discretion and depends on his/her experience. The reopened SFA might be sufficient for appropriate perfusion of the lower leg.

So what evidence exists that PTA is the correct treatment for this case? Level I evidence does not exist, but the consensus is that short lesions of less than 7 cm should be treated in this way [6]. The indication should be the same as for open surgery, namely critical limb ischaemia but also disabling claudication, provided that the patient's quality of life would be very much improved by an increased possibility to walk.

Whether PTA is superior to exercise alone is still under debate [7, 8]. There is, however, no doubt that PTA is more accepted for iliac lesions than for SFA lesions in cases of intermittent claudication. If one accepts PTA, then it has to be clear that recurrence of symptoms is not uncommon, and that the duration of the effect of treatment is variable. Stents may be deployed following iliac angioplasty but are less effective in SFA angioplasty. Improved results are claimed by those who do subintimal angioplasty. The use of stents in the SFA should be reserved at present as a rescue procedure in cases of suboptimal dilation causing dissection or any other damage to the vessel. Controlled studies are needed to define the indications of stenting, in particular with the new generation of drug diluting stents, in the management of peripheral vascular disease.

Complications of PTA include bleeding at the puncture site (about 2 per cent), pseudoaneurysms (0.7 per cent) [9], local vessel damage (see above), and vessel rupture (very infrequent). Bleeding at the puncture site is normally treated with compression, but surgery may be required. Likewise, pseudoaneurysms can be treated by ultrasound-guided compression or thrombin injection. As described, local damage to the vessel with dissection may be treated with a stent; local bleeding caused by rupture commonly does not require any treatment other than careful observation to avoid compartment syndrome due to an expanding haematoma.

When following up patients after PTA, duplex examination can be of value as it can find restenosis long before the ankle blood pressure is reduced, although ABPI should be measured regularly. Other investigations are not required, should no symptoms reappear that demand treatment. **[Q4: B, D]**

All forms on continuing exercise are of importance for all patients after treatment for PAOD and no detrimental effects of cycling should be anticipated; antiplatelet drugs should be administered for secondary prevention of thrombotic events at the treatment site but mainly for coronary and cerebrovascular thrombotic events. Warfarin is not indicated. **[Q5: B, E]**

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16. Lower Limb Claudication due to Iliac Artery Occlusive Disease

Fabien Koskas and Marcus J. Brooks

A 63-year-old hypertensive man presented in 1990 with a history of pain developing in his left calf and thigh after walking 100 metres. During the preceding 3 months, following the introduction of a beta-blocker for newly diagnosed hypertension, the distance he could walk at a "normal" pace had reduced from 200 metres. The pain ceased almost immediately after stopping walking and appeared again after the same interval. A systemic enquiry was unremarkable. He was noted to be an active and life-long heavy smoker. Clinical examination revealed a diminished left femoral pulse and absent left popliteal and pedal pulses. The abdominal aorta and right leg pulses were normal. No bruits were audible in the abdomen or groins.

Question 1

Which of the following would be part of your initial management of this patient?

A. Smoking cessation advice.

B. Enrolment of the patient in a supervised exercise programme.

C. Stopping the beta-blocker.

D. A prescription for warfarin.

E. A prescription for aspirin.

Simple advice was given on the importance of exercise as there was no supervised exercise programme available. The patient managed to stop smoking completely. He returned to the clinic after 3 months and his symptoms had not improved. As the patient was shortly to be retiring and was an enthusiastic hunter he was very keen for any intervention that might relieve his claudication.

Question 2

How would you proceed with your management now he has returned?

A. No further intervention.

B. Drug treatment with cilostazol.

C. Obtain arterial imaging to better define the lesion.

Question 3

Which of the following is *not* an appropriate first line imaging modality?

A. Duplex scan.

B. Contrast-enhanced CT scan (CTA).

C. Contrast-enhanced magnetic resonance angiography (MRA).

D. Digital subtraction contrast angiogram (IADSA).

The patient underwent an arterial duplex scan. This scan showed a significant stenosis at the left internal iliac origin and a short but tight stenosis of the proximal external iliac artery. The contralateral iliac system was found to be free from significant disease, as were the femoral, popliteal and crural arteries. Subsequent angiographic images are shown in Fig. 16.1.

Question 4

Which of the following would you consider as possible interventions?

A. Aorto-bifemoral bypass graft.

B. Left aorto-uni-iliac bypass graft.

C. Right femoral to left femoral cross-over graft.

D. Percutaneous transluminal angioplasty via a right femoral puncture.

E. Percutaneous transluminal angioplasty via a left femoral puncture.

A percutaneous transluminal angioplasty was performed from the left groin using a 7-mm balloon. The left leg pulses were restored by the procedure. The patient noticed that his claudication disappeared.

Question 5

Which of the following statements describe the optimal follow-up for this patient?

A. Low-dose subcutaneous low-molecular-weight heparin for 3 months.



Fig. 16.1. Images from the digital subtraction angiogram showing **a** the renal arteries, infrarenal aorta and iliac bifurcation, **b** stenotic lesions at the origin of the left internal iliac artery and in the left external iliac artery, and **c** an oblique projection of the left iliac system. 🕮

B. The patient is seen regularly, for review of his claudication and control of his risk factors.

C. Serial duplex scanning to detect recurrent stenosis before symptoms occur.

D. The patient is discharged from follow-up.

Eleven years later the patient returned complaining of recent onset of erectile dysfunction and the return of his left calf claudication.

Question 6

What is the likely aetiology of this man's erectile dysfunction?

In the intervening 10 years the patient had resumed smoking and had undergone a coronary artery bypass graft for unstable angina. Two years following the bypass his angina had recurred. A coronary angiogram showed that two of three vein grafts had occluded, and that his left ventricle function was poor (28 percent ejection fraction). On examination his left femoral pulse was weak, the distal pulses were absent in the left leg and a soft bruit was heard over the right femoral artery. The patient insisted on being relieved from his symptoms "no matter the risks" as he had married a woman much younger than him. Another angiogram was requested, one image from which is shown in Fig. 16.2. The distal run-off (not shown) was preserved in both legs.

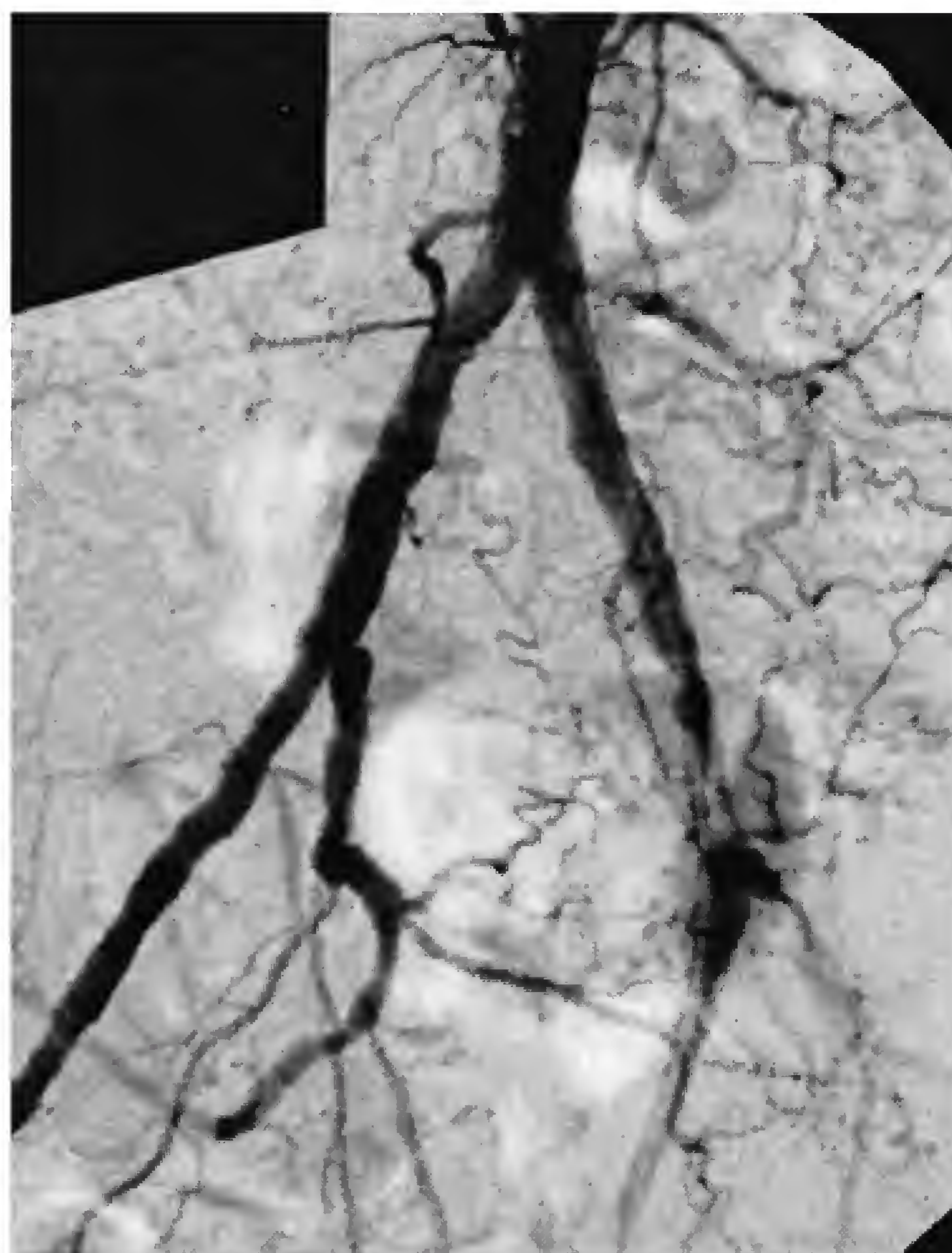


Fig 16.2. Angiogram performed for investigation of the patient's erectile dysfunction and recurrence of left calf intermittent claudication.

Question 7

Which of the following is now the preferred intervention?

A. Aorto-bifemoral bypass graft with revascularisation of both internal iliac arteries.

B. Left aorto-uni-iliac bypass graft with revascularisation of the left internal iliac.

C. Right femoral to left femoral cross-over graft.

D. Percutaneous transluminal angioplasty.

E. No intervention – claudication and impotence are a good way of protecting the heart.

A percutaneous approach was used; a hydrophilic guidewire passed easily through the lesion and both the external and internal iliac lesions were treated endovascularly (Fig. 16.3).

Commentary

The majority of patients with peripheral vascular disease smoke [1]. Cessation of smoking slows the rate of progression of peripheral vascular disease and reduces the risk of cardiac morbidity and mortality [2]. The prescription of nicotine replacement therapy is of benefit in patients who find it difficult to quit [3]. The benefit of exercise for relieving the symptoms of intermittent claudication has long been recognised [4]. The type and frequency of exercise to yield maximum benefit has now been examined in a systematic review and Cochrane Collaboration Overview; advice alone is of little benefit but supervised exercise programmes (achieving maximal walking distance for at least 30 minutes three times a week) can achieve a 150 percent increase in walking distance or 6-minute increase in walking time [5, 6]. A systematic review failed to show any association between beta-blockers and worsening claudication [7]. If the beta-blocker is stopped another antihypertensive agent, such as a calcium channel blocker or ACE inhibitor, should be substituted for control of hypertension, as treating hypertension reduces the stroke risk by 38 percent, cardiovascular risk by 14 percent and peripheral vascular events by 14 percent [8]. A systematic review by the Anti-platelet Trialists Collaboration has proven the benefit of 75–1500 mg aspirin daily in achieving a 25 percent reduction in the risk of death, stroke or myocardial infarction [9]. A post-hoc subgroup analysis of patients with peripheral vascular disease in the CAPRIE trial showed additional benefit for clopidogrel [10]. The additional benefit is small (196 patients on clopidogrel to prevent one death) and not justified except for the 20 percent of patients who are aspirin intolerant. There is no evidence of benefit from warfarin [11]. It is also important to start the patient on statin therapy as this intervention has been shown to achieve an equivalent reduction in morbidity and mortality to aspirin [12, 13]. **[Q1: A, B, E]**

The patient returns having modified his risk factors and is no better. His claudication is affecting his quality of life. The options for management are persistence with unsupervised exercise, drug treatment or intervention. Cilostazol is the only

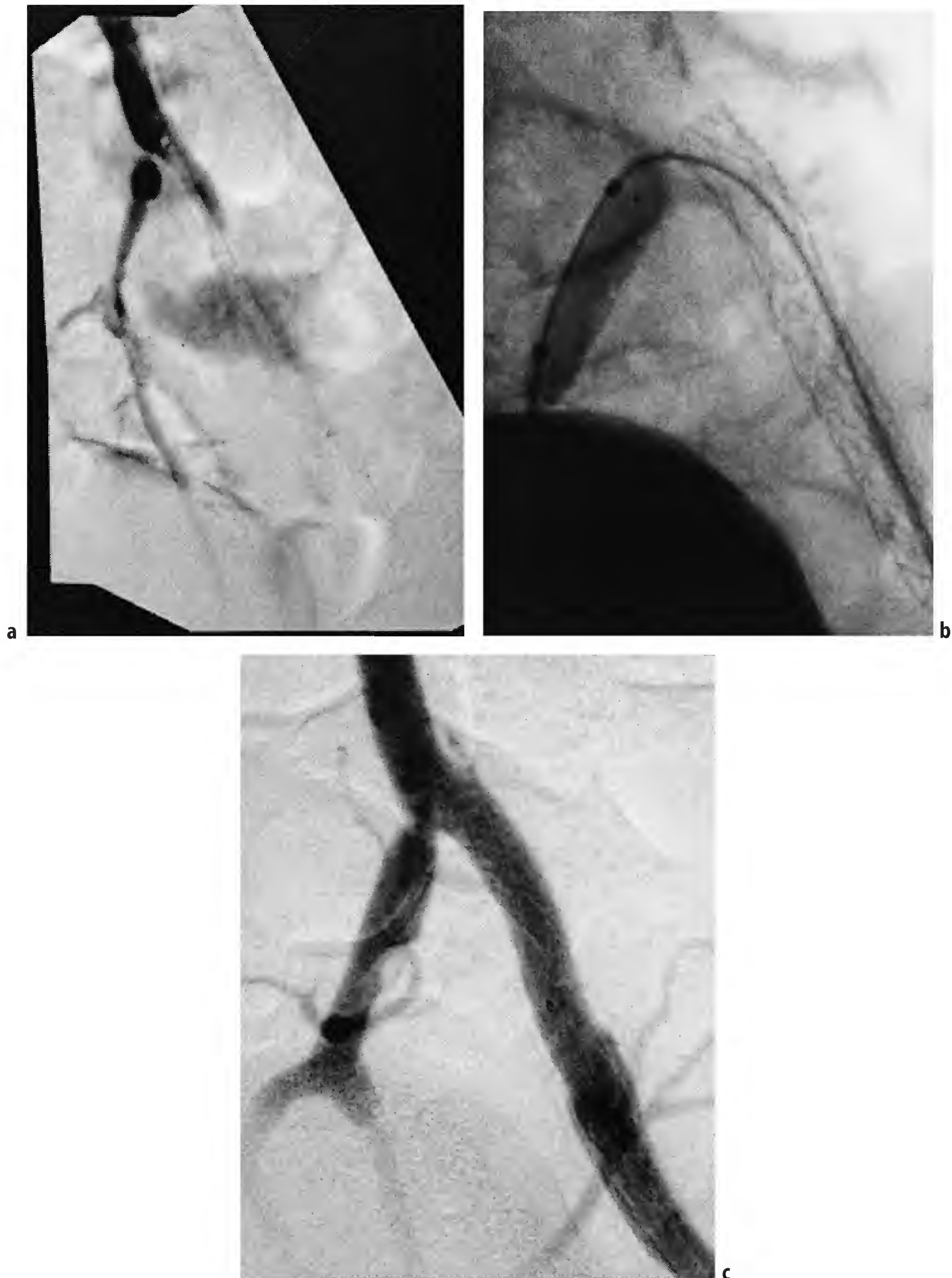


Fig 16.3. Images from the second procedure showing **a** passage of a hydrophilic guidewire across the occluded external iliac artery, **b** an excellent technical result from angioplasty with stent placement in the external iliac artery with the wire now positioned in the internal iliac artery and **c** a completion angiogram.

drug shown to be effective at relieving the symptoms of intermittent claudication in a small randomised trial [14, 15]. However, it is expensive and the effect is short-lived. Intervention can only be considered once the anatomy of the underlying stenosis is known. As the presenting symptom is intermittent claudication and the patient has a weak left femoral pulse with normal right leg pulses we suspect a single level left iliac stenosis. It was decided to image the lesion. **[Q2: C, (B)]**

The optimal imaging of aortoiliac lesions is dependent on the facilities available. It is preferable to first obtain non-invasive images to allow the approach to a lesion to be planned, ensure the appropriate equipment is available and obtain the appropriate patient consent . Duplex scanning has become a useful tool for non invasive evaluation of aortoiliac occlusive disease [16]. However, duplex in the aortoiliac segment is highly dependent on patient's body habitus and experience of the operator. A helical multi-detector row (32 or 64 detectors) CT scanner can provide high-quality cross-sectional images of the aorta, iliac arteries and even arteries down to the feet. CT scans have the advantage to the surgeon of familiarity and show calcified vessel walls. The disadvantages of CTA are the risk of contrast-induced nephropathy, patient exposure to ionising radiation and the time it takes to reformat the images [17, 18]. Contrast-enhanced magnetic resonance angiography (MRA) can also image the aortoiliac segment down to the feet. It is the investigation of choice in patients at risk of contrast-induced renal impairment. In a comparison of CTA and MRA in imaging the aorta and iliac segments, sensitivity and specificity for the detection of lesions were equivalent. CTA took longer to reformat and report; a greater proportion of patients expressed a preference for CTA [19]. MRA is contraindicated in patients with pacemakers and ferromagnetic intracranial aneurysm clips. Intra-arterial digital subtraction angiography now has a limited diagnostic role in the aortoiliac segment. Angiography is invasive and is only performed if artefacts from previous implants (i.e. stainless steel stents) degrade three-dimensional imaging, if direct pressure measurements across a stenosis are required or, as in this patient, as the first stage of an invasive procedure following non-invasive imaging. **[Q3: D]**

The left internal iliac origin and mid-third external iliac artery lesions are TransAtlantic Inter-Society Consensus (TASC) type A lesions [20]. The TASC consensus on the management of type A aortoiliac lesions (Recommendation 32) was for endovascular intervention. Surgical options, endarterectomy or bypass, are reserved for longer stenoses (5–10 cm) or occlusions [20]. The reported primary technical success of angioplasty of type A lesions is 98–99 percent with 60–80 percent patency at 5 years [21]. The 5-year patency of open procedures is slightly better, 90 percent for aorto-bifemoral bypass, but the patient is exposed to the risks of death (2–3 pre cent), erectile dysfunction and graft infection [22]. It is a matter of personal preference whether a left or right percutaneous approach is used for the angioplasty as the lesion is mid-way between the aortic bifurcation and inguinal ligament. **[Q4: D or E]**

The optimal management of patients following angioplasty has not been evaluated in randomised control trials. The risks to the artery are thrombosis, myointimal hyperplasia and disease progression. All patients should already be on an antiplatelet agent. Patients are formally heparinised during the procedure and for this short stenosis this is probably adequate. There is no evidence that post-procedure low-molecular-weight heparin, or for that matter any pharmacological agent (e.g. ticlopidine), is of benefit. Routine graft surveillance has been shown to improve the secondary patency of infra-inguinal vein bypass grafts [23]. Surveillance has not been evaluated following iliac angioplasty. As myointimal

hyperplasia and disease progression both occur, it appears prudent, if not mandatory, to follow up patients. This can be done using clinical examination, arterial duplex or ankle brachial pressure index (ABPI) measurement. Clinical follow-up is cost-effective and is a good way of enforcing a tight control of risk factors. **[Q5: B]**

Erectile dysfunction in this setting is probably due to arterial insufficiency resulting from progression of bilateral iliac occlusive disease as the patient has a new bruit in the right groin. The association of erectile dysfunction with aortoiliac occlusive disease was first described in 1814 by Robert Graham [24]. However, it was Rene Leriche who in 1940 in Paris operated on a 29-year-old truck driver "who for two years had been suffering from claudicatio intermittens with severe cramps in the leg musculature already after a few hundred meters of walking, and cramp pains also at night. The last weeks before the operation he complained of not being able to complete an intercourse, as both erection and ejaculation was disturbed" [25]. **[Q6:** Leriche syndrome**]**

The patient has suffered disease progression in the intervening years. He has developed a very tight stenosis of the left internal iliac artery, a stenosis or the right internal iliac artery origin and complete occlusion of the left external iliac. The left external iliac lesion is classified as a TASC type C lesion [20]. The consensus in 1990, when the TASC guidelines were drawn up, was that definitive recommendations on how to treat such lesions must await more convincing evidence. This situation has not changed. The risks of open aortoiliac bypass surgery and endarterectomy have already been discussed. Remote iliac endarterectomy using Moll ring strippers avoids an abdominal approach and pelvic dissection, has good published technical success rates (88–92 percent), and 3-year patency just below that of open endarterectomy (60 percent) [26, 27]. A potential development for the future is laparoscopic aortoiliac surgery [28]. In this patient a femoral-femoral cross-over graft is not advisable because contralateral lesions may impair the graft inflow and because this procedure would not address the internal iliac stenoses. Had the cardiac antecedents not been present, direct bilateral surgical antegrade revascularization of the lower limbs and one or both internal iliac arteries would have been an excellent solution. However, in the context of unreconstructable coronary artery disease and poor left ventricular function, such a solution is too invasive and carries the risk of potentially lethal cardiac morbidity. On the other hand, surgical abstention, although not without justification, seems exaggerated because quality of life is often as important as its length among middle-aged and aged patients. **[Q7: D]**

Stenting is generally reserved for the management of lesions with a high risk of primary failure (i.e. eccentric calcified plaque), primary failure (residual stenosis greater than 50 percent or greater than 10 mm Hg pressure gradient), dissection or distal embolisation [29]. In this patient, stents were placed because of the recurrent occlusion of the external iliac and residual stenosis in the internal iliac artery. The patient recovered from his claudication, as well as his impotence, without any significant cardiac morbidity and is still doing well 4 months after the procedure.

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17. Erectile Dysfunction due to Aortic Disease

Ralph G. DePalma

A 60-year-old married man presented with sudden onset of erectile failure about 1 month previously. This occurred in the absence of critical life changes, psychological stress, or known intercurrent illness. Initially, the patient was seen by a general practitioner and treated with sildenafil in incremental doses up to 150 mg. This had only a minor effect: ejaculation was maintained but erections remained insufficient for penetration.

Risk factors consisted of mild hypertension controlled by an angiotensin converting enzyme (ACE) inhibitor, cigarette smoking (discontinued 1 month previously), and a history of an infrarenal abdominal aortic aneurysm diagnosed 8 months before. Ultrasound had measured an abdominal component 3.6 cm in diameter compared with a suprarenal aortic diameter of 3 cm.

The patient was started on intracavernous injection of prostaglandin E1 25 μg/ml, which allowed intermittent functional status. However, he reported intermittent failure with this treatment.

Because of this complaint and the previous history of aneurysm, computed tomography (CT) scans and aortography were obtained. The CT scan revealed an infrarenal aneurysm 4.5 cm in diameter and a 2.1-cm aneurysm of the right common iliac artery. Aortography revealed bilaterally patent internal iliac arteries. The diameter of the left common iliac measured 1.8 cm. Penile brachial indices were 0 on the right and 1.0 on the left. Penile pulse waves on plethsymography exhibited delayed upstroke and sine wave forms.

Question I

Which of the following statements regarding impotence is incorrect?

A. Most cases respond to medical treatment, i.e. intracavernous injection or oral therapy.

B. Chronic smoking may render the patient refractory to medical treatment.

C. Impotence may be due to atheroembolism into penile arteries.

D. Impotence may relate to stretching of the nerves by aneurysm.

E. Impotence may be due to pelvic arterial occlusive disease.

Because of the findings of enlargement of the aneurysm and evidence of penile brachial occlusion on the right, a decision was made to operate.

Question 2

What are the techniques for operation under these circumstances?

Question 3

Describe nerve-sparing dissection for aortoiliac involvement and maintenance of internal iliac flow.

Question 4

Which of the following statements regarding the management of abdominal aortic aneurysms in impotence is/are correct?

A. Aortoiliac bypass beyond involved external iliac arteries usually preserves erectile dysfunction.

B. In patients older than 65 years, erectile dysfunction usually improves after aneurysm repair.

C. Concomitant sympathetic nerve injury might result in retrograde ejaculation.

D. An operation that achieves both nerve sparing and preserves internal iliac flow will offer the best chance of continued function.

At operation, the aortic aneurysm was measured at 4.5 cm and contained scanty clot. However, there was a 2-cm aneurysm at the junction of the common and external iliac arteries medially. This pocket contained loose atheromatous debris. Potentially, this related to the absent penile Doppler signal on the right.

Postoperatively, the patient had subjective improvement after reconstruction, reporting spontaneous erectile function for several months. He eventually continued to have satisfactorily regular intercourse using a vacuum constrictor device. There were no changes in the Doppler signals; the plethsymographic wave forms improved in amplitude but not form. The right penile Doppler signal remained absent. The patient functions well at 48 months and appears satisfied with this result. Prevention of further embolisation of the aneurysmal debris possibly prevented further deterioration of erectile function. Penile arteries can also be examined with duplex ultrasonography after intracavernous injections of a vasoactive agent.

Question 5

Are the results of surgery better with direct penile artery bypass for cases of small-vessel disease in young men or for large-vessel occlusive disease in men aged 50 years or older?

Commentary

Impotence (erectile dysfunction) is defined as the persistent or repeated inability for at least 3 months' duration to attain and/or maintain an erection sufficient for satisfactory performance in the absence of an ejaculatory disorder such as premature ejaculation [1]. Erectile dysfunction is a symptom, not a disease; it has a variety of causes. With the availability of effective oral drugs [2], and the knowledge that a minority of men require vascular surgery, treatment usually begins with medical therapy [3, 4]. However, diagnosis and therapy are interrelated [5]. In this case, due to the abrupt onset of dysfunction, failure to respond to medical therapy [6], and a finding of large-vessel disease, the decision was made to investigate the patient further [7]. The usual mechanism of erectile dysfunction in large-vessel disease is occlusion, not stretching of the nerves. **[Q1: D]**

The operation preferred by this author is transabdominal with preservation of the sac of the aneurysm and nerve-sparing dissection at the aortic bifurcation. In this case, the right iliac limb of the graft was passed through the iliac sac and anastomosed to the junction of the external and internal iliac arteries in healthy tissue. The inferior mesenteric artery was suture ligated from within the sac [8]. **[Q2] [Q3] [Q4: D]**

Postoperative treatment for erectile dysfunction should be discussed with the patient and partner before surgery .In this case, recovery of function was probably due partially to embolisation of the right penile arteries, but the patient can now function with a vacuum constrictor device. Among 1094 men with the chief complaint of impotence, only 17 were found to have large-vessel aortoiliac disease as a major cause [9]. This type of presentation of erectile dysfunction is uncommon [10], but it is important as the results of intervention can be better than small-vessel bypass [11] surgery in well-selected cases [12].

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18. Bypass to the Popliteal Artery

Jeannie K. Chang, Keith D. Calligaro and
Matthew J. Dougherty

A 62-year-old overweight postal worker presented with complaints of cramps in his right calf. He stated that this reproducible pain occurred each time he walked 50 yards and resolved upon sitting down. He denied tissue loss or rest pain. His past medical history was significant for hypertension, hypercholesterolemia and tobacco use, as well as coronary revascularization.
On physical examination, he had bilateral carotid bruits, normal heart examination, and a strong right femoral pulse, but absent popliteal and pedal pulses. His left lower extremity had a saphenectomy scar. Both extremities had shiny, hairless skin without ulcerations or gangrene.

Question 1

Which of the following is not an indication for a bypass to the popliteal artery?

A. Mild to moderate intermittent claudication.

B. Non-healing toe ulcer with an ankle brachial index (ABI) of 0.30.

C. Rest pain.

D. Symptomatic popliteal aneurysm, entrapment syndrome, or adventitial cystic degeneration.

The patient's blood pressure and cholesterol levels were controlled well by medication. He lost excess weight, quit smoking, and initiated cilostazol therapy, but to no avail. His symptoms persisted and he was so incapacitated that he was unable to continue delivering the mail.

Arteriography was performed, demonstrating patency of the right iliac arteries but severe occlusive disease of the superficial femoral artery. There was reconstitution of the popliteal artery with two-vessel run-off. The patient consented to a femoropopliteal bypass procedure.

Question 2

The conduit yielding the best long-term patency for this bypass is:

A. Dacron.

B. Autologous vein.

C. PTFE.

D. Umbilical vein.

E. Cryograft vein.

Question 3

A distal cuff or patch is most likely worthwhile for which type of bypass?

A. Femoropopliteal above-knee reversed vein graft.

B. Femorotibial in situ vein graft.

C. Femoropopliteal above-knee PTFE.

D. Femorotibial PTFE.

E. Femoral-femoral PTFE cross-over graft.

Femoropopliteal bypass was performed with in situ greater saphenous vein to the below-knee popliteal artery. There was resolution of the patient's claudication, and he was able to return to work. Unfortunately, he became lost to follow-up, and 2 years later he returned with complaints of recurrent claudication in his right lower extremity. Neither popliteal nor pedal pulses were palpable. Duplex ultrasonography and arteriography demonstrated several sites with elevated velocities, suggestive of two moderate focal stenoses in the proximal half of his bypass graft as well as a severe narrowing at the distal anastomosis.

Question 4

What are the treatment options for a failing graft?

A. Aspirin therapy.

B. Percutaneous transluminal angioplasty (PTA).

C. Laser-assisted angioplasty and atherectomy.

D. Amputation.

The patient was taken to the operating room, where a longitudinal incision was made through the distal portion of his vein graft and popliteal artery. Under fluoroscopy, balloon angioplasty of the proximal moderate stenoses was performed, with excellent results. Using a small segment of autologous saphenous vein, patch

angioplasty of the distal anastomosis was performed. Completion angiography revealed a widely patent graft, and his distal pulses were again appreciated on palpation. He was able to resume his usual activities and was seen routinely in the vascular clinic.

Question 5

The most useful serial postoperative test to assess graft patency and a possible failing graft is:

A. Arteriography.

B. Pulse volume recordings.

C. Duplex ultrasonography.

D. Ankle brachial index.

E. Magnetic resonance angiography (MRA).

Commentary

Mild to moderate intermittent claudication is not an indication for surgical bypass. Most (approximately 75 per cent) patients presenting with only intermittent claudication have a benign course, remaining stable or improving with conservative measures, such as smoking cessation, weight loss and alteration in diet, graduated exercise programs, and medical treatment of risk factors (e.g. hypertension, hypercholesterolemia, diabetes). Claudication is a strong and independent predictor of mortality, however, and thus concomitant identification of comorbidities such as coronary and cerebrovascular atherosclerotic disease may have significant impact on survival.

Pharmacological therapy may be initiated with rheological agents such as pentoxifylline or cilostazol with variable effect. Antiplatelet therapy is frequently started to prevent cardiac or cerebrovascular complications. Only a minority (10–20 percent) of patients require surgical reconstruction, and few (3–6 percent) ultimately progress to major amputation [1].

Revascularization is reserved for patients with disabling claudication or evidence of critical ischemia manifest as acute motor or sensory loss, chronic tissue loss or rest pain. Other less common etiologies for lower-extremity ischemia may cause femoropopliteal occlusion and are occasionally indications for surgical revascularization. **[Q1: A]**

Long-term patency rates are highest when autologous vein is used as conduit. If the greater saphenous vein is not available, then lesser saphenous vein, femoropopliteal vein, or upper-extremity veins may be acceptable alternatives. The advantages of in situ vein bypass grafting include the preservation of the vein's nutrient supply and the better size match of the proximal and distal artery to the proximal and distal vein. Using reversed vein grafts, however, avoids the endothelial trauma of valve lysis. Although at times somewhat conflicting, the literature does not support the superiority of one technique over the other for femoropopliteal bypasses.

The use of human umbilical vein [2] or cryopreserved vein has also been described with varying success. The latter may be a potential alternative to prosthetic grafts if autologous vein is unavailable, but in below-knee revascularization, cryopreserved vein has demonstrated the tendency for aneurysmal degeneration and poor long-term patency [3].

Prosthetic grafts in the suprageniculate bypass have demonstrated patency rates that are comparable with those for autologous vein [4]. The type of prosthetic graft is less important than the age of the patient or the size of the conduit [5]. The patency of prosthetic grafts to infrageniculate arteries, however, is significantly worse than that of autologous vein. Further, the use of composite prosthetic and autologous vein does not seem to improve long-term patency compared with pure prosthetic grafts [6].

Finally, there have been reports of endovascular treatment of femoropopliteal atherosclerotic disease, including percutaneously inserted covered stents [7] and prosthetic grafts introduced through a femoral arteriotomy and anchored distally with stent deployment [8]. Long-term patency with these techniques remains to be evaluated. **[Q2: B]**

Intimal hyperplasia occurs frequently at the distal anastomosis when a prosthetic graft is used for an infrainguinal bypass and compromises its survival. Modifications to improve long-term patency include various vein cuffs and patches. Using these techniques theoretically improves compliance match between the prosthetic material and the artery at the distal anastomosis. The reduction in turbulence minimizes the trauma to the arterial endothelium and decreases its proliferative response.

The Miller cuff was studied in a prospective randomized study to determine its potential benefit in improving the patency rate of distal supra- and infrageniculate femoropopliteal polytetrafluoroethylene (PTFE) grafts. Although no difference was noted in above-knee bypasses with or without vein cuff, a statistically significant improvement in patency was observed in below-knee procedures [9]. Similarly, the Taylor patch has been reported to improve patency of infrageniculate bypasses [10]. **[Q3: D]**

Salvage of a bypass graft in the early postoperative period may include strategies such as thrombectomy and revision of technical errors. These errors include graft kinks, retained valve leaflets, intimal flaps, and residual arteriovenous fistulas in an in situ graft.

Recently, percutaneous endovascular techniques such as balloon angioplasty have been utilized with increasing frequency but with equivocal results. Focal lesions (less than 20 mm) are more amenable to catheter-based techniques than are more diffuse stenoses, but even these favorable lesions may recur. Laser angioplasty and atherectomy, however, have not been shown to be beneficial in the preservation of failing grafts.

Thrombolysis may be considered for patients who present with sudden and recent onset of symptoms attributable to bypass graft occlusions. For patients with chronic graft occlusion, a new bypass graft provides improved clinical outcome, but in acute graft occlusion, thrombolysis may improve limb salvage and reduce the magnitude of the subsequent surgical procedure [11].

For short-segment stenoses, patch angioplasty or interposition within an existing vein graft with autologous or prosthetic material may be performed to preserve a bypass to the popliteal artery. Although technically simpler and requiring less autologous material, patch angioplasty has inferior results when compared with inter-

position [12]. Longer-segment stenoses are preferably treated with interposition or jump graft around the area of narrowing. Failing these strategies, however, the creation of an entirely new bypass may be required.

Amputation is reserved for tissue loss or ischemic pain without possible vascular reconstruction. Long-term survival of patients requiring major amputation is poor. **[Q4: B]**

Early graft failure (i.e. within the first 30 postoperative days) is most likely the result of a technical error, hypercoagulability, poor distal run-off, or postoperative hypotension. In addition to avoidable technical errors, early graft failure may be secondary to endothelial trauma, the use of imperfect conduit, or poor surgical judgement with regard to the adequacy of inflow or outflow.

Graft failure within the first few years of surgery is usually attributable to intimal hyperplasia. Subsequent graft failure is most frequently secondary to progression of atherosclerotic disease.

It is vital to identify a failing graft before complete occlusion to preserve the patency of the graft. Lower-extremity revascularization can be salvaged with simple interventions if lesions leading to intimal hyperplasia and hemodynamic compromise can be identified before graft thrombosis. Revision of stenotic lesions in a failing but nonoccluded graft results in superior patency when compared with revision of similar lesions in an occluded graft. It also leads to fewer amputations and subsequent revisions. Additionally, the repair of a failing graft is less costly than emergent revision of a failed graft or of amputation [13].

Postoperative duplex ultrasonography detects correctable abnormalities early, precludes the need for angiography in many cases, and markedly improves assisted primary patency of vein bypass grafts. Recommended surveillance includes initial ABI and duplex studies at 1 week, followed by evaluations at 3, 6, 9, 12, 18, and 24 months, then annually thereafter. High-grade stenoses can be identified and corrected before thrombosis occurs. Criteria for the diagnosis of a failing graft include monophasic signals, peak systolic velocity (PSV) less than 45 cm/s throughout the bypass, any PSV greater than 300 cm/s, or a PSV ratio across a stenosis of greater than 3.5 [14]. **[Q5: C]**

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19. Chronic Critical Limb Ischemia

Enrico Ascher and Anil P. Hingorani

An 85-year-old male with a history of diabetes, hypertension, hypercholesterolemia, coronary artery bypass, and active tobacco use presented with a gangrenous right first toe. The patient stated that he had no history of trauma to the area, and complained of rest pain in the foot. The patient had been in otherwise good health since his coronary artery bypass 12 years ago. On physical examination, the patient was in no physical distress. The patient had a well-healed median sternotomy scar. Auscultation of the heart revealed a regular rate without any murmurs. He was obese. Abdominal examination revealed no palpable masses. The patient had bilateral femoral and popliteal pulses but no pedal pulses. The patient had bilateral, well-healed scars from the greater saphenous vein harvest sites. The right gangrenous toe was dry without any evidence of infection.

Question 1

Which of the following statements regarding chronic lower-extremity ischemia are wrong?

A. If the patient refuses any intervention, then anticoagulation alone may be helpful.

B. The contralateral asymptomatic lower extremity should also undergo angiography as there may be severe atherosclerotic disease there as well.

C. The treatment options remain unchanged if the patient presents with only rest pain, ischemic ulcer or claudication.

D. The patient cannot undergo revascularization without contrast arteriography as there are no other alternatives.

The patient's arterial duplex demonstrated moderate distal right superficial femoral artery disease. The ankle brachial indices (ABIs) and pulse volume record-

ings demonstrated findings consistent with moderately decreased perfusion at the calf level and severely decreased perfusion at the ankle and transmetatarsal levels. The cardiac review of systems was unremarkable, and a persantine thallium obtained 6 months ago revealed no perfusion defects. Electrocardiogram (ECG), chest X-ray and routine preoperative blood tests were normal. Venous duplex mapping revealed inadequate veins (sclerotic and too small) in the bilateral upper and lower extremities.

Question 2

Preoperative medications/lifestyle changes that should be added to the patient's regimen to reduce his overall cardiovascular risk based upon randomized prospective data include:

A. Aspirin.

B. A statin.

C. Angiotensin-converting enzyme inhibitors.

D. Tobacco cessation.

E. A beta-blocker.

Percutaneous angiogram of the right lower extremity demonstrated moderate right distal superficial femoral artery stenosis with distal occlusion. The popliteal appeared to be severely diseased with occlusion of the tibioperoneal artery and proximal anterior tibial artery. The mid-anterior tibial artery reconstituted and ran down to the dorsalis pedis artery. No other vessels appeared to be adequate.

Question 3

What type of options would you consider for this lower extremity?

A. Below-knee amputation.

B. Digital amputation.

C. Tibial bypass with expanded polytetrafluoroethylene (ePTFE) with a venous interposition or fistula.

D. Tibial bypass with cadaveric vein.

E. Sympathectomy.

F. Chelation therapy.

G. Subintimal angioplasty

The patient underwent a successful bypass with ePTFE to the anterior tibial artery and did stop smoking after the procedure. The patient's toe underwent auto-amputation and the rest pain has resolved. He was followed up 2 years after the procedure with a patent bypass.

Question 4

What is the patient's long-term prognosis in terms of mortality, graft patency, and limb salvage after successful bypass?

A. The long-term mortality, patency, and limb salvage are about 20 percent and therefore are so poor that no intervention should be made.

B. The mortality and patency are 50 percent at 4–5 years. The limb salvage is 70 percent at 4 years. If the patient has a reasonable life expectancy and functional status, he should undergo the revascularization.

C. The mortality, patency, and limb salvage rates are irrelevant in this age group.

Question 5

Which patients would you consider to be inoperable? What treatment options may be offered to this subset of patients?

Commentary

Indications for revascularization to the tibial vessels are limited to ischemic ulcers, gangrene, and rest pain. The long-term patency of the bypass is affected directly by continued tobacco use, and the patient should be urged to stop smoking. Anticoagulation plays no role as the sole management of this patient. Even though the patient may have asymptomatic contralateral disease, there is no role for further investigation. Angiography may be used to visualize both inflow and outflow sites. In general, the most distal available inflow site is utilized to shorten the length of the graft. Time-delayed imaging may be required to visualize the calf and foot arteries because of reduced flow. The use of magnetic resonance angiography (MRA) has proven to be beneficial in identifying patent lower-extremity arteries, particularly in view of the recent advances in imaging software and hardware [1–3]. Finally, high-resolution duplex imaging has now become a viable alternative for visualization of inflow and outflow sites with the added advantages of cost reduction, fewer complications associated with angiography, and the ability to identify the least calcified artery segment [4–8]. However, both MRA and duplex imaging should be used only as preoperative imaging modalities after they have been validated at each center. **[Q1: A, B, C, D]**

Increasing focus on the perioperative and long-term management of patients with peripheral arterial disease has identified that all the factors listed in Question 2 can significantly reduce the incidence of cardiovascular events in these patients. These data have been supported by large multicenter randomized prospective trials [9] Therefore, it becomes incumbent on the vascular surgeon to also consider these as part of the treatment plan when evaluating a patient with peripheral arterial disease. **[Q2: A, B, C, D, E]**

Evolution of vascular surgery techniques in the past decade, combined with the availability of an adequate venous conduit, has permitted a liberal and aggressive approach to salvage ischemic limbs caused by advanced atherosclerosis. This

approach is epitomized by the construction of arterial bypasses to the terminal branches of tibial vessels [10]. However, significant numbers of patients continue to face the threat of a major amputation because of insufficient vein necessary to perform a totally autogenous bypass to one of the infrapopliteal arteries. In these cases, less durable grafts made of prosthetic material must be used if limb salvage is to be attempted. Accordingly, several adjunctive techniques have been designed in an attempt to improve the poor patency results achieved with prosthetic bypasses. These include the administration of immediate and chronic anticoagulants [11], the construction of a vein patch or cuff at the distal anastomosis to prevent occlusion by intimal hyperplasia [12, 13], and the creation of an arteriovenous fistula to increase graft blood flow in high-outflow-resistance systems [14, 15]. Despite initial enthusiasm, the results using cadaveric vein have been poor and resulted in its very limited use [16, 17]. **[Q3: C]** If the popliteal artery had been not as diseased, an attempt at subintimal angioplasty with angiography or with duplex guidance may also be considered [18, 19].

The expected long-term mortality of this patient is 24–50 percent at 4–5 years and is due mostly to myocardial ischemia [20]. The expected patency of these techniques is 50–60 percent at 3–4 years [20–23]. The expected limb salvage rates are 70–80 percent at 3–4 years [20–23]. **[Q4: B]**

Based on these data, we would suggest that there is no role for amputation or sympathectomy in this particular patient. However, if the patient had prohibitive cardiac risks, had nonreconstructable disease, or was already so neurologically impaired that the limb was not of any utility to the patient, then observation, primary amputation, hyperbaric oxygen therapy or perhaps experimental protocols involving angiogenesis factors may be in order. **[Q5]**

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20. Popliteal Artery Entrapment

Luca di Marzo and Norman M. Rich

A 26-year-old female presented with a 6-year history of cold foot, paraesthesia and cramping in both legs after intensive physical training. She was a recreational bodybuilder and complained of her symptoms mostly after sporting activity. Symptoms subsequently became more severe, with cramping requiring 20 minutes to release after sport.

Question 1

What is the presentation of cases with popliteal artery entrapment?

A. The patient is often sporty with muscular calves.

B. The patient often complains of rest pain or necrosis.

C. The patient often complains of mild symptoms with paraesthesia, cold foot and cramping after intensive physical training.

D. Venous complaints are often encountered.

E. Symptoms due to arterial embolisation are often present.

The patient smoked 20 cigarettes a day. Her past medical history included pancreatitis when she was 12 years old and tonsillectomy when she was 19 years old. On physical examination, she appeared healthy, with both legs appearing athletic. Lower-limb pulses were normal, but bilateral pedal pulse reduction was noted after calf muscle contraction. A popliteal artery entrapment (PAE) was therefore suspected, and the patient was sent for noninvasive vascular evaluation. Doppler and colour Doppler showed normal posterior tibial and popliteal recordings, with signal disappearance on both legs during calf muscle contraction. Doppler examination was conducted with the patient supine recording the posterior tibial artery during manoeuvre (Fig. 20.1). Colour Doppler was performed, with the patient prone, and the sample volume placed in the popliteal artery. Muscular contraction of the calves showed an arterial occlusion on colour flow imaging (Fig. 20.2).

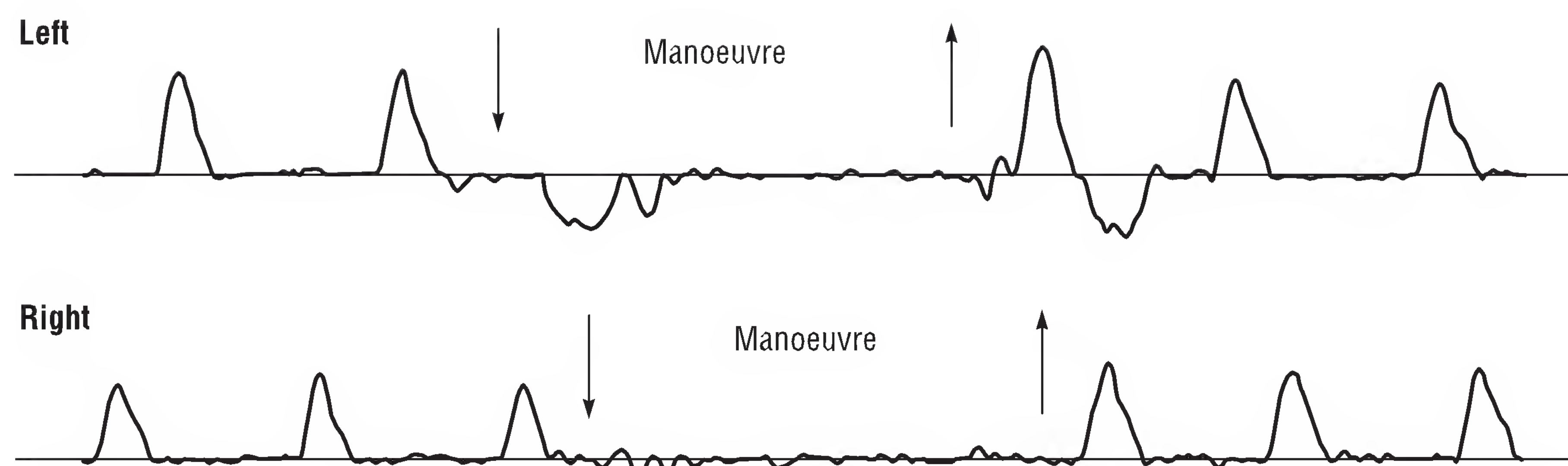


Fig. 20.1. Continuous-wave Doppler recording the posterior tibial artery during manoeuvre.

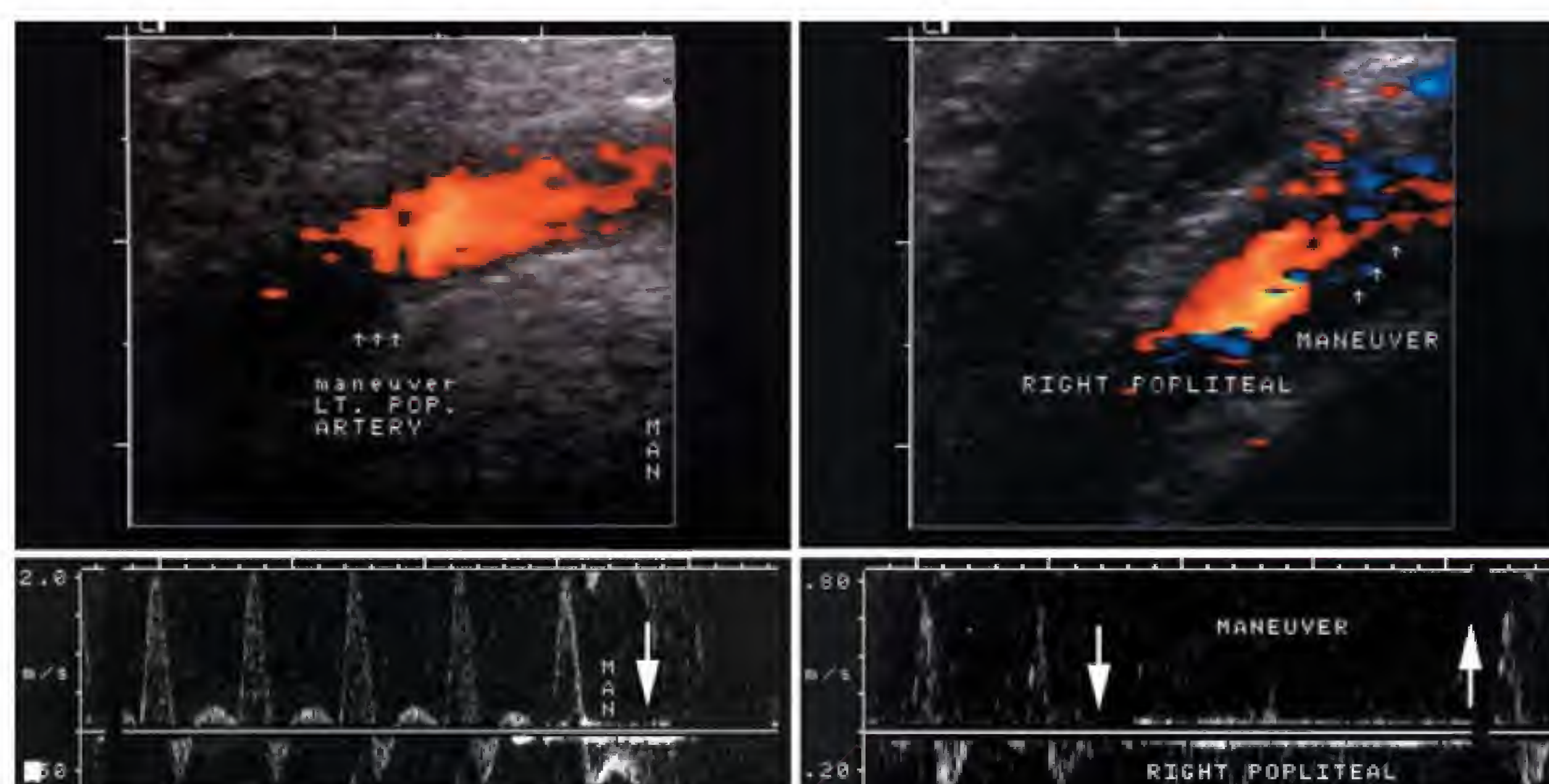


Fig. 20.2. Colour Doppler during muscular contraction of the calves, showing arterial occlusion.

Diagnosis of bilateral PAE was made. Arteriography was conducted to confirm the diagnosis: it showed normal popliteal arteries, with right severe stenosis and left occlusion during calf muscle contraction (Fig. 20.3). Magnetic resonance angiography (MRA) was attempted, which demonstrated bilateral popliteal occlusion during manoeuvre (Fig. 20.4).

Question 2

How will you make the diagnosis of PAE?

A. Doppler can detect PAE.

B. Arteriography is only carried out preoperatively to confirm results of ultrasound scans.

C. MRA may be diagnostic in the hands of an experienced practitioner.

D. Duplex scanning can detect PAE.

E. Computed tomography (CT) scanning can detect PAE.



Fig. 20.3. Arteriography showing normal popliteal arteries, with right severe stenosis and left occlusion during calf muscle contraction.

Question 3

Which of the following statements regarding angiograms of a patient with PAE are correct?

A. Normal angiograms at rest are often encountered in entrapments.

B. The angiograms show an occlusion or severe stenosis during calf muscles contractions.

C. Three-vessel run-off is often encountered in PAE.

D. An arterial occlusion is encountered in PAE diagnosed at a late stage.

E. A post-stenotic aneurysm may be encountered.

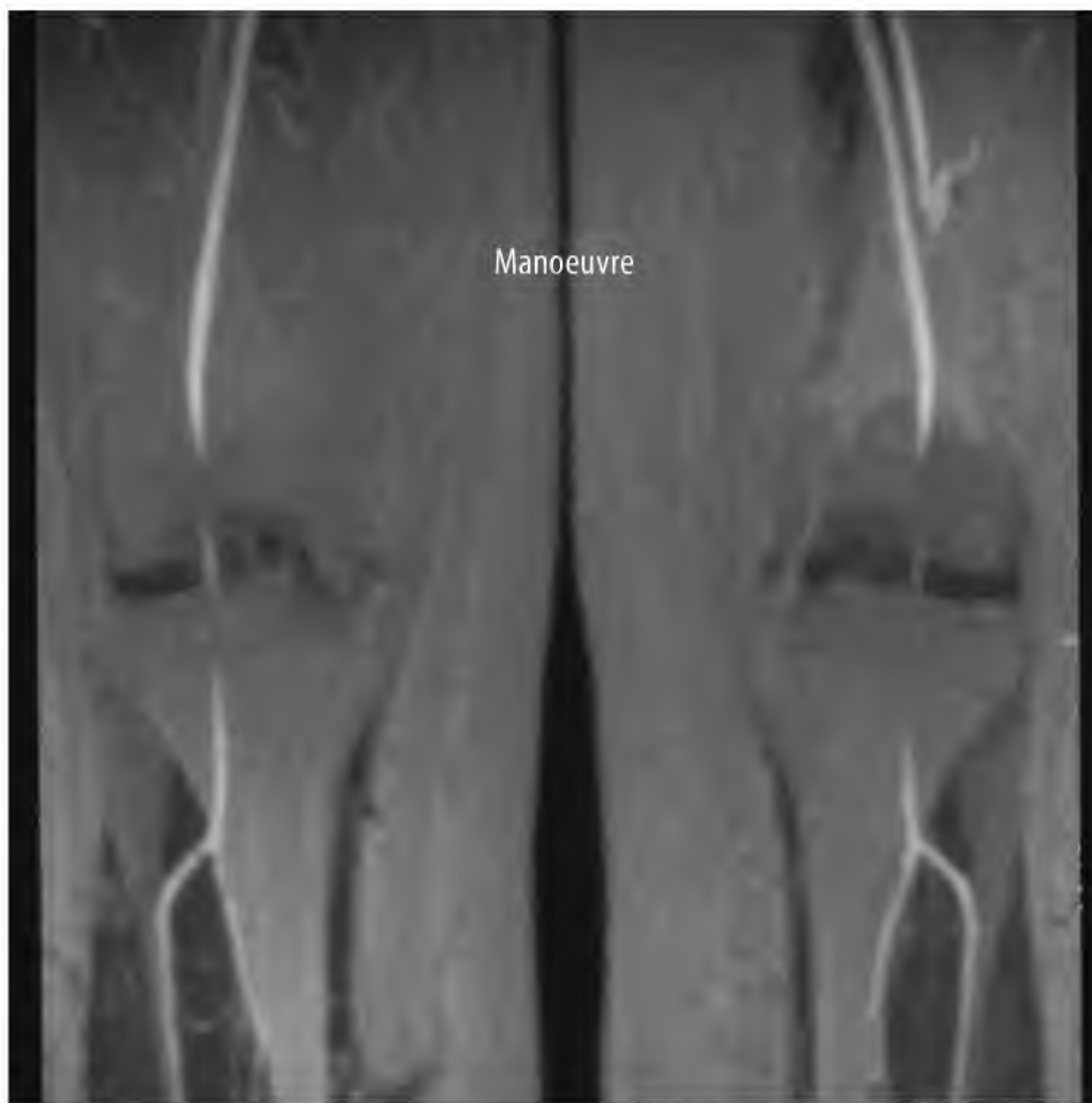


Fig. 20.4. MRA demonstrating bilateral popliteal occlusion during manoeuvre.

The patient was considered for bilateral surgical treatment. A posterior approach to the popliteal fussa was made through a Z-shaped incision. The medial gastrocnemius muscle had a large accessory head with a lateral and cranial insertion, causing bilateral compression of the popliteal artery and vein. This head was resected on both legs, without any need for muscular reconstruction.

Question 4

Which of the following statements regarding the treatment of PAE are correct?

A. Musculotendineous sectioning is the treatment of choice in patients with a normal popliteal artery.

B. Vascular reconstruction should be limited to cases with stable arterial impairment.

C. If vascular reconstruction is planned, then the use of autologous vein is mandatory.

D. The posterior approach is recommended to expose all the structures causing compression.

E. The structure causing PAE must be sectioned completely, as incomplete sectioning may cause recurrence.

Question 5

Which of the following statements regarding the incidence of entrapment are correct?

A. The medial gastrocnemius muscle is involved in almost 80 per cent of cases of PAE.

B. Venous entrapment is described more often than arterial entrapment.

C. Venous entrapment is concomitant in 20 per cent of cases of PAE.

D. More than one structure may be the cause of arterial entrapment.

E. Classification of arterial entrapment includes 12 different types.

The postoperative course was uneventful and the patient was discharged 5 days after surgery, returning back to normal activity after 3 weeks. Follow-up demonstrated complete regression of symptoms. Ultrasound examinations (Doppler and colour Doppler) showed normal popliteal flow with negative response to PAE manoeuvres 1 month after surgery. The patient is now doing sport (swimming) again without any further complaints.

Commentary

The first case of PAE was treated surgically in 1959 in a 12-year-old boy complaining of claudication after walking 300 m. At surgical exploration, Hamming [1] at Leyden University in The Netherlands found an occluded artery with an anomalous course medial to the medial gastrocnemius muscle. He transected the muscle and performed a successful popliteal artery thromboendarterectomy.

A previous description of the disease was reported in 1879 by Stuart [2], a medical student at the University of Edinburgh. During the dissection of an amputated leg of a 64-year-old man, he observed the popliteal artery coursing around the medial head of the gastrocnemius muscle and aneurysmal changes in the popliteal artery distal to the point of external muscular compression.

Since than, many case reports have been published. A few authors have published small series [3–6]. Unfortunately, the papers that were collected were missing details and showed poor patient follow-up [7].

In Rome in 1998, the Popliteal Vascular Entrapment Forum was founded. Surgeons from around the world with the greatest experience in this field world were invited as founding members of the forum. Great effort was addressed to collect different series with comparable criteria. The criteria established by the Society for Vascular Surgery (SVS) were reviewed and accepted, with some minor changes. Common opinion was to consider both arterial and venous entrapment as a common disease defined as vascular entrapment. The functional form of

Table 20.1. Classification of popliteal vascular entrapment

Type	Features
I	Popliteal artery running medial to the medial head of gastrocnemius
II	Medial head of gastrocnemius attached laterally
III	Accessory slip of gastrocnemius
IV	Popliteal artery passing below popliteal muscle and medial head of gastrocnemius
V	Primary venous involvement
VI	Variants
F	Functional entrapment

entrapment was discussed. This was first described by Rignault et al. [8] in 1985, and describes cases in which the anatomy of the popliteal fussa is normal. Symptoms are usually caused by hypertrophy of the muscles determining a compartment syndrome [8, 9]. Functional entrapment was included in the classification as type F (Table 20.1).

Popliteal artery entrapment is no longer a rare disease. It is encountered more and more often, particularly in young adults. Athletes practising sports causing hypertrophy of the limb muscles are at higher risk due to an anomalous relationship of the popliteal artery and its surrounding musculotendineous structures. The artery is compressed each time the leg moves, causing peripheral ischaemia during intensive exercise. With time, this intermittent arterial trauma may give rise to stable arterial damage, with occlusion or post-stenotic aneurysm. Early diagnosis and treatment play an important role in limiting surgical treatment to the sectioning of the structure causing the arterial compression. **[Q1: A, C]**

The diagnosis of PAE is based primarily on ultrasound scanning. Both continuous-wave Doppler and colour Doppler are able to detect the presence of an arterial compression due to entrapment. The manoeuvres to be performed are well described and are able to detect suspected cases [7]. Great care should be taken to suspect early cases of PAE in patients complaining of minor symptoms (paraesthesia, cold foot and cramping after intensive physical training). Arteriography is limited to cases with positive ultrasound examinations, and it requires great care in repeating the manoeuvres to confirm the popliteal compression. MRA may be diagnostic, but it needs latest-generation apparatus and the input of a radiologist with great experience in both the disease and the imaging method. At present, MRA cannot be considered a first-choice examination, but it is reasonable to assume that in the future it will substitute angiography in the diagnosis of entrapment. **[Q2: A, B, C, D] [Q3: True A, B, C, D, E]**

Surgical treatment consists of sectioning the musculotendineous structure causing the entrapment. The anomalous structure needs to be sectioned entirely in order to avoid recurrence of the entrapment due to hypertrophy of the remaining anomalous muscle. It is important to remember that complete exposure of the popliteal fussa is obtained through a posterior approach. The medial approach limits the view of the medial gastrocnemius muscle. In our opinion, this exposure should be limited to cases in which the arterial impairment is extended to the tibial vessels and a distal reconstruction needs to be planned. However, early diagnosis allows surgical treatment to be limited to the muscle sectioning, which should be considered the first-choice treatment. When a popliteal severe stenosis, occlusion or aneurysm is present, then an arterial

reconstruction is indicated. In this case, we recommend the use of autologous material to reconstruct the artery. This improves the long-term patency rate. Great effort should be paid for alternative vein preparation when the saphenous vein is unavoidable. **[Q4: A, B, C, D, E]**

The medial gastrocnemius muscle is often the cause of compression. However, more than 20 different anatomical variants have been described, and sometimes multiple and complex structures may be associated with the medial gastrocnemius muscle in causing PAE. The popliteal vein is involved in the compression in 20 per cent of cases affected by PAE. Moreover, isolated popliteal vein entrapment is described with increasing frequency in the literature. **[Q5: A, C, D]**

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21. Adventitial Cystic Disease of the Popliteal Artery

Bernard H. Nachbur and Jon Largiadèr

Case 1

A 49-year-old female presented with a 3-week history of left calf intermittent claudication at 150 m, which had occurred suddenly and without preliminary herald signs. The patient was a nonsmoker and had no risk factors, such as hypertension, diabetes or hyperlipidaemia. She was engaged in regular sporting activity, playing tennis all year round and skiing in the winter. She thought at first that it might be a strained muscle and would subside spontaneously. This did not happen and she sought medical advice.

At clinical examination, the popliteal and pedal pulses of the left leg were barely palpable and were absent after exercise. Angiological examination of the right leg was normal. The ankle systolic pressure at the right side was 128 mm Hg with a slight rise to 132 mm Hg after exercise. On the left side, ankle systolic pressure at rest was 88 mm Hg with a post-exercise reduction to 58 mm Hg. On duplex sonography, a 5-cm long polycystic swelling surrounding the left popliteal artery was found to be the cause of occlusion of the popliteal artery. The superficial femoral artery and the infrapopliteal arteries showed no trace of atherosclerotic disease. Ultrasonography demonstrated that the content of the cyst was clear and homogeneous. No other cause for popliteal occlusion was found.

Question 1

What is the aetiology of this condition?

An angiogram (Fig. 21.1) showed a 3-cm long subtotal occlusion of the proximal popliteal artery suggesting medial compression, an eccentric form of occlusion reminiscent of an hourglass stenosis (scimitar sign). The top frame of the cross-section of the computed tomography (CT) scan performed at the same time shows an adventitial cyst of approximately 1.5 cm in diameter adjacent to the artery, actually within the arterial wall.



Fig. 21.1. Hourglass-shaped subtotal occlusion of the middle portion of the popliteal artery (scimitar sign) caused by compression by a cyst in the arterial wall, which can be seen in the top panel of the cross-section of the CT scans.

Question 2

Which of the following statements regarding adventitial cystic disease are correct?

A. It affects only the popliteal artery.

B. It can occur elsewhere, such as in arteries near the hip, wrist or ankle joints.

C. It presents with initial signs of acute occlusive disease.

D. It usually begins with intermittent claudication.

E. It can be elicited by loss of pedal pulses during hyperextension of the leg.

F. The cyst is calcified and contains atheromatous material.

G. The cyst contains a viscous gelatinous fluid.

The popliteal artery was laid free posteriorly through a S-shaped popliteal incision. The arterial wall contained a cyst filled with a gelatinous mucoid yellowish substance. The occluded arterial segment was resected and replaced by interposition of a segment of saphenous vein. Fig. 21.2 shows the popliteal artery before and after surgery with complete normalisation of patency.

Case 2

A 49-year-old woman complained of sporadic episodes of intermittent claudication of varying intensity [1]. At times, she could walk freely; at other times, after physical exercise with bending of the knee, intermittent claudication would occur after walking distances of 200–300 m. Angiography revealed only discrete semilunar narrowing of the middle portion of the popliteal artery, as shown in Fig. 21.3 (scimitar sign). At the time of this examination, the patient had hardly any complaints.

Question 3

Adventitial cystic disease of the popliteal artery can be diagnosed reliably by:

A. Duplex coloured sonography.

B. Injection of indium^{111} and scintigraphy.

C. The semilunar sign (scimitar sign) or hourglass sign at angiography.

D. A meniscus-shaped proximal occlusion at angiography.

E. T2-weighted magnetic resonance imaging (MRI).

F. Systolic bruit in the hollow of the knee.

G. Intravascular ultrasound imaging.

H. CT scanning.



Fig. 21.2. Popliteal adventitial cyst before and after segmental resection and interposition of a segment of autologous vein.

Question 4

What are the treatment options?

The popliteal artery was laid free posteriorly through a popliteal incision. The arterial wall was surrounded by a 5-cm long polycystic tumour in the centre of which was a 3-mm wide stem that could be followed to the knee joint. A fine probe was introduced for injection of contrast medium. The cyst took the appearance of a Baker cyst, which was filled with a jelly-like yellowish mucoid substance. The cyst



Fig. 21.3. Angiography of the popliteal artery, with a discrete semilunar deformity (arrow pointing to the scimitar sign). At the time of this angiography, the patient was in momentary clinical remission.

was found to be lying in the outer layers of the adventitia and was removed easily without causing any damage to the artery itself (Figs 21.4 and 21.5).

The varying clinical presentation of intermittent claudication in this case can be explained by pressure changes occurring within the cyst during different physical activity [1]. Histologically, the wall of the cyst consisted of collagenous connective tissue covered on the inside by a single interrupted or several layers of cuboid cells

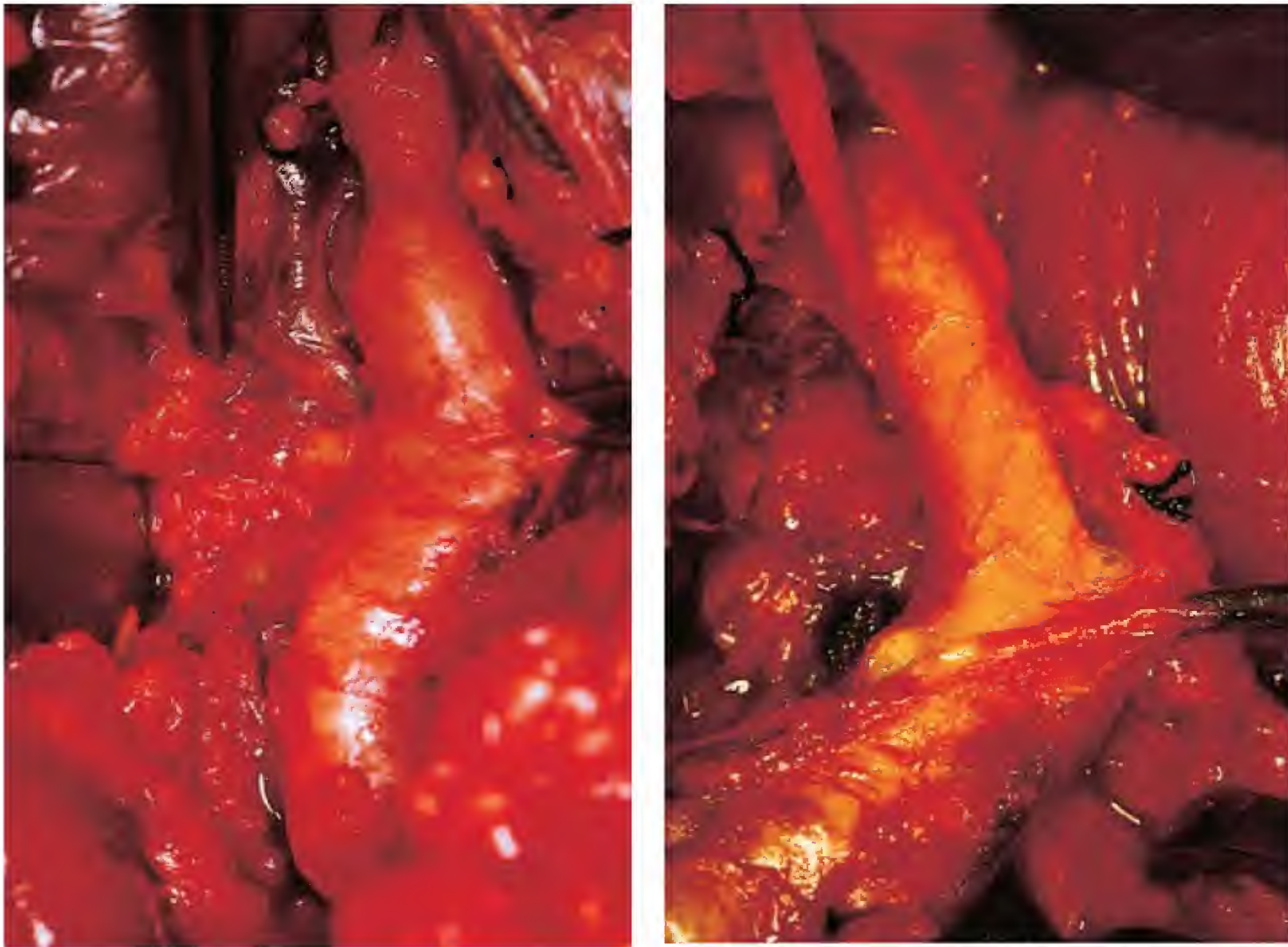


Fig. 21.4. *(left)* The whole extent of the 6-cm long cyst surrounding the popliteal artery.

Fig. 21.5. *(right)* The perivascular cyst being resected, with the artery remaining intact.

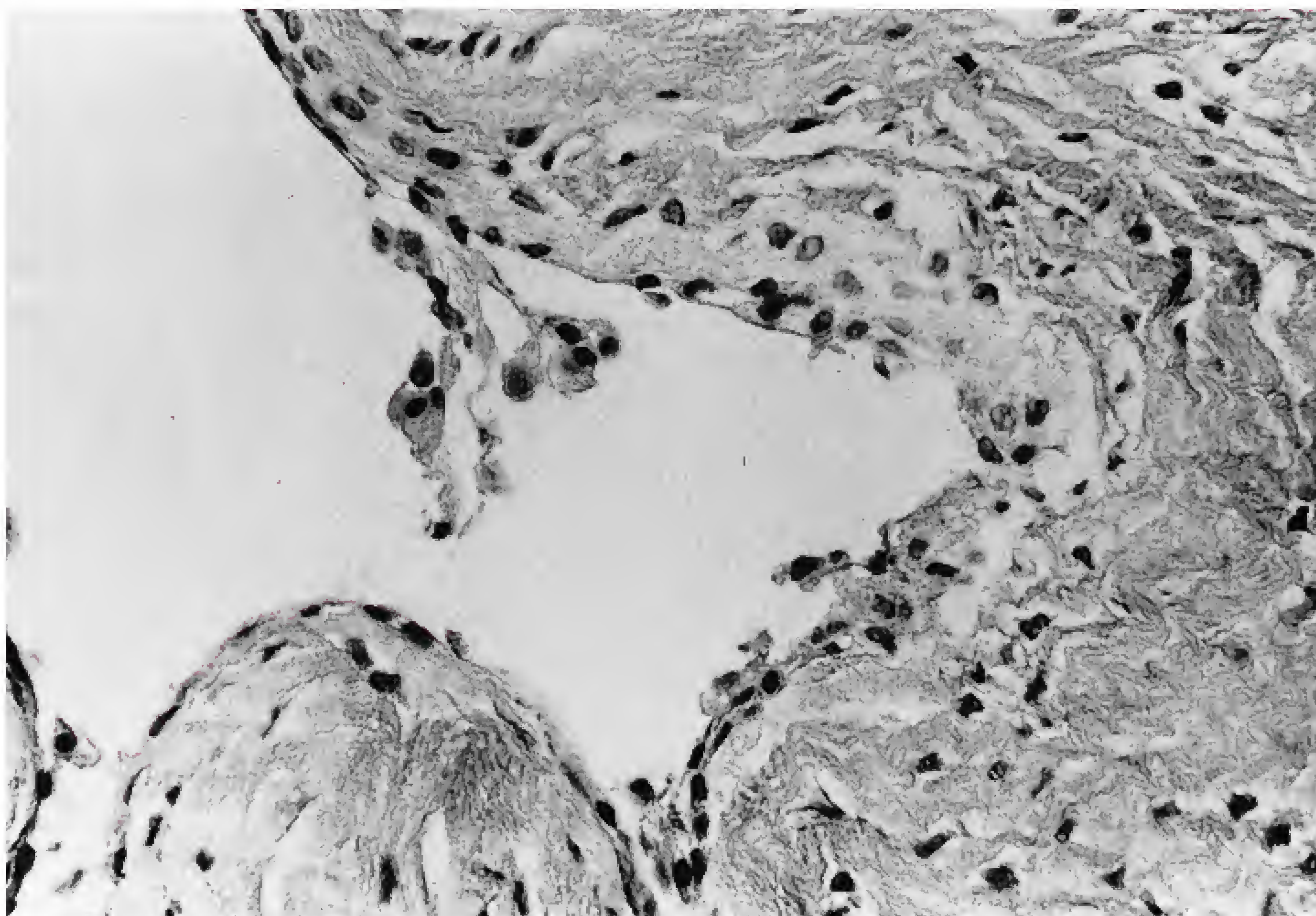


Fig. 21.6. The wall of the cyst covered on the inside by a single interrupted or several layers of cuboid cells akin to synovial mesothelium.

akin to synovial mesothelium [2] (Fig. 21.6). The stem connecting with the knee joint had a similar structure. The lumen of both cyst and stem contained viscous basophil fluid; they are therefore best likened to ganglions.

Commentary

Trauma has been ruled out overwhelmingly on the grounds that the disease would be seen predominantly in people engaged in competitive sports: this is not the case. All cases of adventitial cystic disease reported in the literature have occurred in nonaxial vessels during limb differentiation and development. It is therefore postulated that during limb bud development, cell rests derived from condensations of mesenchymal tissue destined to form the knee, hip, wrist or ankle joints are incorporated into the nearby and adjacent nonaxial vessels from vascular plexuses during the same stage of development, and in close proximity to the adjacent condensing joint structures [3]. It is postulated further that these cell rests are then responsible for the formation of adventitial cystic disease in adult life, when mucoid material secreted results in a mass lesion within the arterial or venous wall [3]. Fig. 21.7 shows a row of cross-sections of a resected and totally occluded popliteal



Fig. 21.7. Cross-section through an artery with a large adventitial cyst and compression of the arterial lumen of the resected popliteal artery.

segment. In this case, the cyst is clearly in the midst of the arterial wall and does not appear to be located in the adventitia.

According to the hypothesis of Levien and Benn [3], popliteal adventitial cystic disease manifests itself in adults. Early cases manifest in the third decade, but most cases occur in the fourth and fifth decades; it occurs less frequently in later stages of life [4]. The male : female ratio is about 5 : 1. In summary, there is little doubt that popliteal cystic disease is congenital. **[Q1]**

Popliteal adventitial cysts are located mostly in outer levels, i.e. in the adventitia of the popliteal artery, but they may also occur in the common femoral artery adjacent to the hip joint along the iliofemoral axis, in locations near the elbow or the wrist, and in veins [5]. A total of 45 extrapopliteal localisations have been described. These extrapopliteal locations account for 20–25% of all cases of adventitial cystic disease. Carlsson et al. [6] have also observed adventitial cystic disease in the common femoral artery. **[Q2: B, D, G]**

Because of the sometimes varying degree of intermittent claudication or occasional disappearance of symptoms, the disease can be mistaken for a popliteal entrapment syndrome. Noninvasive techniques have vastly improved diagnosis. Duplex coloured scanning followed by T2-weighted MRI now appear to be the best choice. Both methods are capable of visualising the cyst surrounding the popliteal artery and ruling out the popliteal entrapment syndrome [4]. Koppensteiner et al. [7] have shown that intravascular ultrasound imaging can reliably identify adventitial cystic disease as well. Digital subtraction angiography is necessary to define the degree of stenosis or the length of occlusion. Stenotic lesions have an hourglass appearance or present with a semilunar impression (the scimitar sign) [4]. **[Q3: A, C, E, G, H]**

The treatment options depend on the degree of stenosis and whether the popliteal artery is occluded. In the case of total occlusion, most authors have resorted to total resection of the affected popliteal arterial segment with interposition either of autologous vein or ring-enforced polytetrafluoroethylene (PTFE) grafts. The initial success rate is reportedly almost 90 per cent [4].

If the cyst lies within the adventitia and surrounds and compresses the artery without having given rise to total occlusion, as in our second case, then the artery does not have to be resected if the cyst can be removed entirely [1]. Partial removal of the cyst is thought to bear the risk of recurrence [1]. If a connecting stem usually accompanied by a small collateral artery is present, then this should be resected at the level of the knee capsule to avoid recurrence [2]. The initial success rate in 68 cases treated accordingly is 94 per cent [4]; in our own experience, it was successful in case 2 described above [1].

There is the possibility of resecting only part of the artery, e.g. the medial vascular aspect that bears the cyst, and then replacing the wall defect with a vein patch. This approach has been used in a small number of patients, with success in three of four cases [4]. Percutaneous transluminal angioplasty (PTA) has been performed just once, and failed. PTA should therefore probably be discarded as an treatment option.

An interesting series of seven cases has been reported by Do et al. [8]. They forwarded a 14-gauge needle with real-time ultrasonic guidance transcutaneously directly into the cyst and aspirated its contents in cases presenting with stenosis only (but not in the presence of total occlusion). This was carried out on an outpatient basis, with a 100 per cent success rate. Follow-up colour duplex sonography performed between 1 and 32 months after the procedure showed no recurrent stenosis [8].

While the method of percutaneous aspiration of a popliteal cyst guided by ultrasonography is appealing because it can be done on an outpatient basis and mini-inva-

sively, the question of recurrence is not settled since the cyst remains in place; hence the capacity to form mucinous substance remains and with it the possibility of recurrence. Although Do et al. know of no recurrence in their cases followed up for 1–32 months, there is a definite need for a more systematic long-term follow-up, which should be conducted in all cases in which the cyst has not been removed by resection.

There is the occasional report of percutaneous clot lysis of occluded popliteal arteries followed by aspiration of the contents of the cysts. This method was reported by Samson and Willis [9] to be successful, but its reliability has not been proven by others. There is hardly a valid contraindication against surgical removal of an occluded popliteal segment in the presence of occlusion, and this is probably the method of choice that offers the greatest chances for complete recovery.

Finally, there are reports of spontaneous resolution of the popliteal cysts [10, 11]. It must be assumed, therefore, that occasionally cysts can burst or their contents escape into the periarticular space. This mechanism has been surmised by Soury et al. [10].

In conclusion, the treatment of choice remains surgical resection, either of the cyst alone if it surrounds the artery or of the occluded segment if total occlusion and appositional thrombosis has occurred. In this case, vein graft interposition should be performed. In expert hands, percutaneous transluminal aspiration has been shown to be efficacious. **[Q4]**

Acknowledgement

Special thanks go to Professor Jon Largiadèr, who offered the documentation of the two patients operated on by him at the University Hospital of Zurich.

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22. The Obturator Foramen Bypass

Andries J. Kroese and Lars E. Staxrud

A 62-year-old man presented with a 2-week history of continuous pain in the left lower abdomen radiating to the groin. For several weeks, he had complained of general malaise, including tiredness and poor appetite, and diarrhoea once or twice per day. His general practitioner palpated a pulsating, tender mass in the left groin and referred him to the department of vascular surgery at the nearby university hospital. Three years previously, he had been operated upon with a Dacron aorto-bifemoral bypass for critical ischaemia and intermittent claudication in the left and right lower limbs, respectively.

On admission, the patient was in a relatively good general condition, although his body temperature was 38.5°C, pulse rate was 96 bpm, and blood tests showed an elevated sedimentation rate, C-reactive protein (CRP) and leucocyte count. Palpation of the left iliac fossa was slightly painful. The inguinal swelling was covered by erythematous skin and was estimated to be approximately 4 cm in diameter.

Question 1

What is the most likely diagnosis at this stage?

A. False aneurysm.

B. Infected Dacron graft.

C. Lymphadenitis.

D. Incarcerated inguinal or femoral hernia.

E. Incarcerated obturator hernia.

Based on the clinical signs and symptoms, treatment with broad-spectrum antibiotics was started.

Question 2

Which of the following investigations should be carried out to confirm the diagnosis, and in what order?

A. Duplex scanning.

B. Arteriography.

C. Computed tomography (CT) scanning with aspiration of perigraft fluid for Gram staining and culture.

D. Magnetic resonance imaging (MRI).

E. Leucocyte-labelled scintigraphy.

F. Surgical exploration.

Ultrasonography revealed that the Dacron graft and femoral arteries were not pathologically dilated but that the anastomotic site was surrounded by fluid. Some of this perigraft fluid was aspirated and was found to contain coagulase-negative staphylococci (CNS). Antibiotic treatment was adjusted accordingly.

Question 3

Vascular graft infection in the groin may be treated without resecting the graft itself when there is:

A. Less than 2.5-cm diameter false aneurysm formation.

B. An infected anastomosis, but without bleeding.

C. A thrombosed graft.

D. No septicaemia.

MRI and CT scanning revealed that only the left limb of the bifurcation graft was infected, most likely only in the groin, involving the site of the anastomosis.

Question 4

What treatment options, in addition to antibiotics, are available for the management of an infected vascular graft in the groin?

A. Excision with or without a revascularisation procedure.

B. Repeated extensive wound debridement, and insertion of gentamicin mats.

C. Debridement, skin closure, and insertion of a closed irrigation system.

D. Debridement and muscle flap transposition.

E. None; use long-term antibiotic treatment only.

Since the proximal limit of graft infection could not be ascertained, it was decided to operate on the patient with a partial graft resection. Because the indication for primary operation had been critical ischaemia due to multilevel atherosclerotic disease, revascularisation was planned. Therefore, a preoperative angiography was performed, which showed signs of progressive atherosclerosis as compared with previous angiograms. The proximal part of the left superficial femoral artery was occluded, whereas the distal part was patent. Of the crural arteries, only the posterior tibial was patent. The profunda femoral artery was patent but peripherally stenotic. In the right lower extremity, the superficial femoral artery was occluded, but the profunda artery and three crural arteries were patent, although partially stenotic. Based on these findings, an obturator foramen bypass (OFB) on the left side was planned.

Under general anaesthesia, an 8-mm ring-reinforced polytetrafluoroethylene (PTFE) graft was implanted as an OFB between the proximal part of the limb of the previously implanted Y-graft and the distal superficial femoral artery. During the same operation, the distal part of the infected graft was resected.

Question 5

What is the most common indication for an OFB procedure?

A. Infected femoral (false) aneurysm.

B. Revascularisation with extensive local trauma.

C. Tissue scarring in the groin subsequent to radical tumour surgery, radiation or burns.

D. Sciatic artery aneurysm exclusion.

E. Infection confined to the distal part of an aortofemoral bypass graft.

Question 6

Describe briefly how you would perform an OFB procedure.

After a hypotensive period on the first postoperative day, the left lower limb showed clinical signs of increased ischaemia. Blood pressure at the ankle was 60 mm Hg and the ankle brachial pressure index (ABPI) was 0.4 – slightly lower than preoperatively. Duplex scanning could not rule out a technical defect of the OFB, for example kinking. Therefore, an angiography via the right groin was performed, which did not show any major technical defects (Fig. 22.1). Subsequently anticoagulation therapy with warfarin was started.

Question 7

What is the least frequent complication of an OFB?

A. Urinary bladder injury.

B. Injury of the obturator nerve and blood vessels.

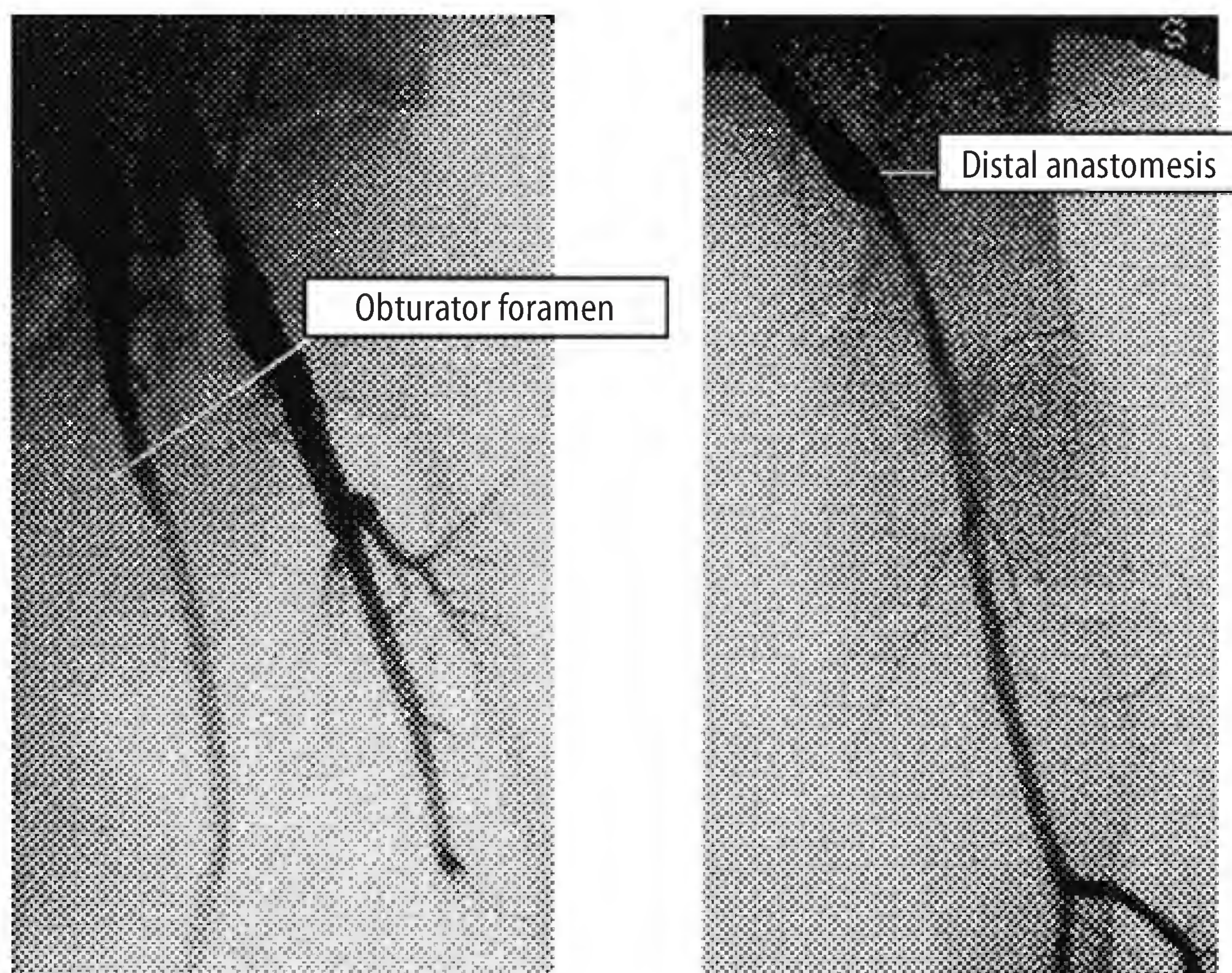


Fig. 22.1. Postoperative angiography of an OFB without signs of technical defects.

C. Kinking of the graft due to erroneous transmuscular tunnelling.

D. Infection of the obturator graft.

E. Bleeding, thrombosis.

F. Injury of the internal iliac artery.

The further postoperative course was uneventful. Two weeks later, the patient was discharged with complaints of claudication in the left lower extremity and a walking distance of approximately 50 yards. Oral antibiotics were to be continued for 3 months and anticoagulation indefinitely.

Question 8

What alternative revascularisation procedures after removal of an infected vascular graft in the groin may be considered?

A. Subintimal angioplasty of the native iliac artery.

B. Semi-closed endarterectomy (ring-stripping) of the iliac artery.

C. Axillofemoral bypass by lateral route.

D. Subvulvular bypass.

E. Subscrotal bypass.

F. Bypass with autologous vein.

Commentary

In patients with a vascular prosthesis anastomosed to the external iliac or common femoral artery, presenting with a painful tumour in the groin, the primary tentative diagnosis should be infected graft. Alternative diagnoses include non-infected false aneurysm, incarcerated inguinal, femoral or obturator hernia, and lymphadenitis. **[Q1: B]**

Preoperative Measures

Even though positive cultures may be lacking, treatment with intravenous broad-spectrum antibiotics, including those against anaerobic microorganisms, are initiated on clinical suspicion of graft infection alone. Late vascular graft infections may be caused by CNS, low virulent bacteria that are often difficult to diagnose by standard techniques [1].

Preoperatively, it is crucial to obtain as much information as possible about the extent of graft infection. Duplex scanning ultrasonography is an appropriate first modality to evaluate perigraft or other groin masses. CT scanning is more effective in the diagnosis of aortic graft infection, especially when combined with aspiration of perigraft fluid for Gram staining and aerobic and anaerobic cultures [2]. MRI can be even more reliable [3]. However, optimal diagnostic accuracy may be obtained by combining CT or MRI with indium-labelled leucocyte scintigraphy [4]. Duplex scanning and arteriography do not play significant roles in establishing the diagnosis of a vascular graft infection, but they are used for diagnosing graft occlusion, false aneurysm formation and anastomotic bleeding, and for planning the revascularisation procedure. In certain cases of inguinal graft infection, contrast sinography may be appropriate to investigate the extent of infection. Finally, when vascular graft infection is suspected despite negative diagnostic tests, surgical exploration of the graft is necessary to detect the presence of perigraft fluid or to confirm whether the graft is incorporated in tissue. It is generally accepted that firm in-growth of surrounding tissue into the vascular prosthesis excludes the presence of graft infection. Although CT scanning and MRI can be very helpful in delineating the boundaries of infection preoperatively, the final judgement concerning the extent of infection is usually made intraoperatively. **[Q2: A, C, D, E, F]**

If only the distal part of the graft is infected, there are several therapeutic options in addition to antibiotics. **[Q3: A, B, D]** If the proximal part of the graft is also infected, then it should be removed entirely. If a revascularisation procedure is warranted, then an extra-anatomic bi- or unilateral axillofemoral bypass may be established, preferably as a first-stage procedure before the entire infected graft is removed.

In the majority of cases, for example if the graft is occluded and the limb is viable, no vascular reconstruction is required [5]. In cases of limited infection, with no signs of anastomotic bleeding or septicaemia, then local treatment without graft resection may be attempted: wound debridement, irrigation, the use of gentamicin-containing collagen mats, and muscle transposition may be alternative ways of treating inguinal vascular graft infections [6]. **[Q4: A, B, C, D]**

If only the distal part of an aortofemoral prosthesis has to be removed, and revascularisation is necessary, then OFB is a very good alternative. It is not a common operation and comprises less than 0.5 per cent of all arterial reconstructions [7].

Since Shaw and Baue [8] introduced this procedure, published results of OFB rarely comprise more than 10 patients [7, 9–13]. However, vascular surgeons should be familiar with its indications and technique when addressing challenging revascularisation problems in a hostile groin.

The Concept of the Obturator Foramen Bypass

The rationale behind this operation is based on creating an arterial conduit from the aortoiliac segment to the superficial femoral, popliteal or deep femoral artery, depending on run-off conditions, while avoiding contaminated, infected or destroyed tissues in the groin. By routing the vascular graft through the obturator foramen, dorsally to the hip joint, in a layer between the adductor magnus and longus muscles, the area of the femoral triangle is circumvented. Autologous saphenous vein has been shown to give satisfying results, reducing the danger of secondary graft infection [14]. However, since the saphenous vein may be too narrow and/or too short, in most cases an externally reinforced Dacron or PTFE graft is used, especially since these conduits offer greater resistance against compression and kinking. Under special circumstances, the obturator bypass can be performed as a cross-over ilioprofunda procedure using the contralateral iliac artery as the inflow site, the graft being routed through the prevesical space of Retzius [15].

The main indication for this procedure (80 per cent of cases) is infection confined to the distal iliac and inguinal part of an aortofemoral bypass graft [1]. Other indications include the need for a revascularisation procedure in cases of infected femoral aneurysm, extensive local trauma [16], tissue scarring in the groin subsequent to radical tumour surgery, and/or therapeutic radiation or burns [17, 18]. The obturator bypass has also been used in rare cases for revascularisation of sciatic artery aneurysm exclusion [19]. **[Q5: E]**

Obturator Foramen Bypass Technique

The patient lies in the prone position, usually with the hip and knee joints slightly flexed, abducted and externally rotated. Some surgeons prefer to have the hip joint overextended a little to facilitate the tunnelling manoeuvre through the obturator foramen. The operation is usually performed under general anaesthesia, sometimes combined with epidural anaesthesia to relieve postoperative pain. In all cases, a urinary catheter should be in place, since urinary bladder injury is a potential danger of this operation. **[Q6]**

If the indication for surgery is an infected prosthetic vascular graft in the groin, then it is an advantage to determine in advance whether a reconstruction is necessary. Thus, the sterile part of the operation, establishing a new vascular conduit, can be done first [1]. The infected groin is sealed off with occlusive drape. Through a longitudinal paramedian incision, the proximal part of the graft is approached transperitoneally. Retroperitoneal access is a good alternative if one is certain that the infection is limited to the inguinal area. The involved graft limb is dissected proximally, close to the bifurcation. Firm incorporation of the graft in the surrounding tissue and a negative Gram stain of perigraft fluid indicate that the proximal part of the graft can be preserved [20]. The graft limb is then transected, and the distal part is closed by sutures and pushed down towards the inguinal ligament.

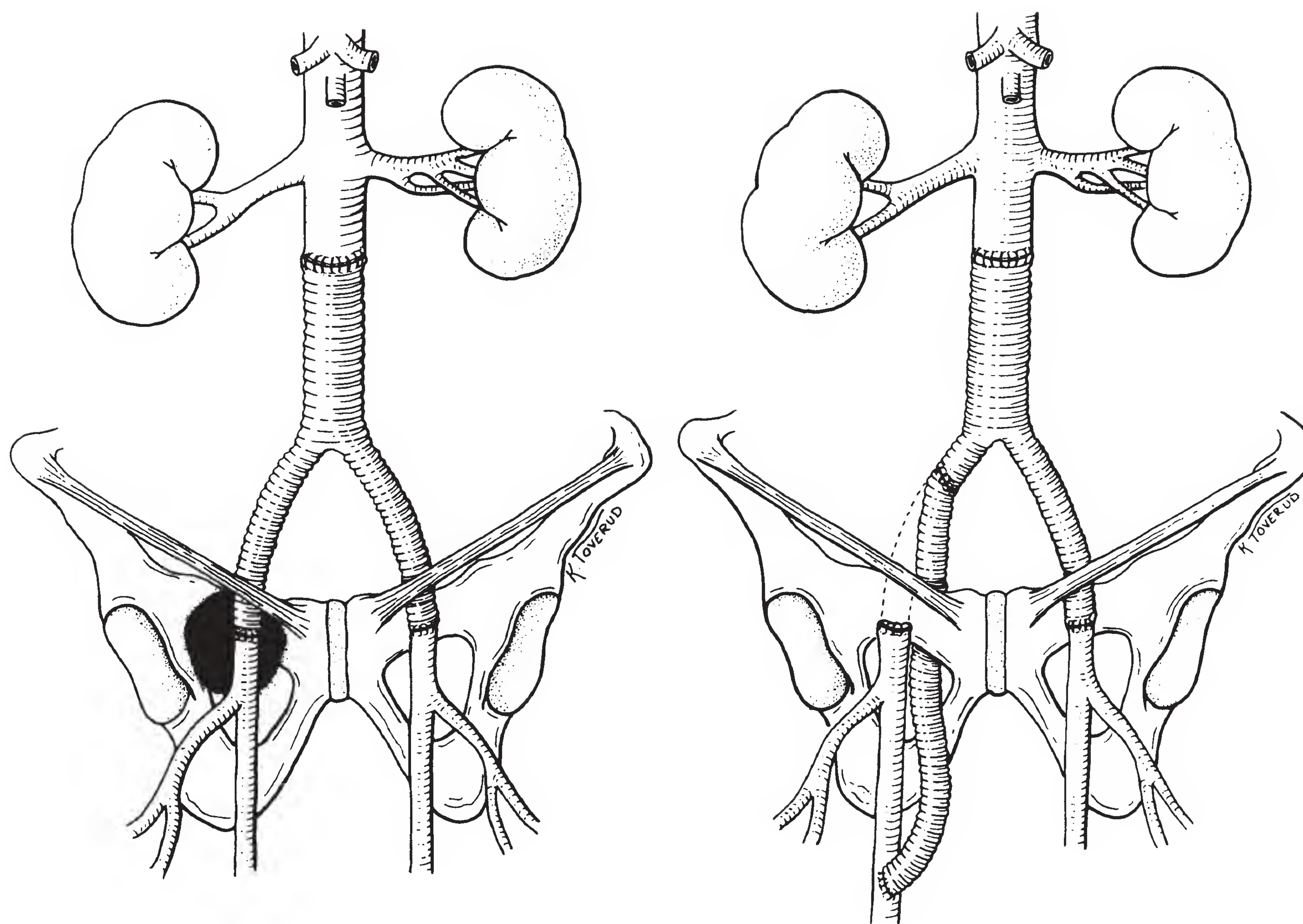


Fig. 22.2. The principle of OFB.

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The overlying peritoneum is oversewn to separate the proximal graft from the infectious area. A ringed PTFE graft of diameter 6 or 8 mm is anastomosed in an end-to-end (Fig. 22.2) or end-to-side fashion to the proximal limb of the bifurcation graft. By careful blunt and sharp dissection, and with the aid of a large-blade self-retaining retractor, the ureter and bladder are identified. The pelvic organs are pushed gently towards the midline, rendering access to the obturator foramen. The sharp edge of the opening in the obturator fascia is usually identified easily by digital palpation on the anteromedial aspect of the foramen. This opening is dilated with long, slim grasping forceps with a blunt tip, taking care not to damage the obturator artery, vein and nerve that curve around the lateral edge of the foramen. Alternatively, other designs of blunt tunnellers can be used. It is therefore prudent to lead the forceps through the foramen bimanually, palpating where the tip of the forceps is to meet the fascial opening. We prefer tunnelling through the obturator foramen from below, in a plane anteriorly to the adductor magnus muscle and posteriorly to the pectineus, adductor longus and brevis muscles. Some surgeons choose to do this manoeuvre from the retroperitoneal space downwards [21]. The PTFE graft may be irrigated retrogradely with heparinised saline to ensure unrestricted flow.

Through an incision in the thigh, medial to the sartorius muscle, the femoropopliteal or profunda femoral artery is exposed for the distal anastomosis, which is usually performed in an end-to-side fashion. The profunda femoral artery is situated anteriorly to the adductor magnus and brevis muscles, covered partially by the adductor longus muscle. By retracting the superficial femoral vessels and the vastus

medialis muscle laterally, a dense fascia between the adductor longus and the vastus medialis is exposed. This fascia is incised, thereby severing the attachment of the adductor longus to expose the profunda vessels. The overlying profunda vein is often divided and ligated to simplify the approach towards the profunda artery [22, 23].

After closing the abdominal and thigh incisions, the patient is redraped and the infected groin is exposed. Swabs are taken for bacterial culture. Necessary debridement is performed, the infected anastomosis is excised, and the femoral artery is closed with a running monofilament suture. The infected graft is removed by withdrawing it under the inguinal ligament from the retroperitoneal space. Finally, the wound is irrigated lavishly before closing it over a suction catheter.

Perioperative complications occur in approximately 7 per cent of cases [8, 12, 24]. Bleeding from obturator vessels can be prevented by adhering to sound surgical principles.

Perforation of the urinary bladder, vagina or sigmoid colon by faulty tunnelling of the graft is a serious complication that may lead ultimately to loss of limb [25, 26].

Since the obturator bypass is threatened by infection, long-term postoperative antibiotic treatment is advised. Although the duration is debatable, a period of 6–12 weeks can usually be agreed upon. Graft thrombosis may lead to severe ischaemic symptoms, and may even threaten the viability of the lower limb, since important collateral vessels in the inguinal region may have been sacrificed during the previous operation. Gluteus muscle necrosis may also compound this critical situation. Therefore, thrombectomy or thrombolysis of the thrombosed OFB graft should be attempted without delay. **[Q7: F]**

The obturator bypass in the management of infected vascular grafts seems to be a valuable procedure [12, 27]. However, long-term results with this operation in terms of patency, limb salvage and survival rates are difficult to evaluate because the studies are usually small and include cases with different indications for obturator bypass. However, the majority of patients suffer from symptomatic peripheral arterial disease. In a review of the literature, perioperative mortality rates varied between zero and 14 per cent. Survival rates after 1 and 5 years were 81 and 61 per cent, respectively. Secondary patency rates for PTFE prostheses at 1 and 5 years were 71 and 52 per cent, respectively. Short-term limb salvage rates up to 76–85 per cent [1] and a 5-year salvage rate of 55 per cent could be achieved [12]. The results depend on the indication for operation and are better in patients without atherosclerosis. In patients with atherosclerosis, graft patency depends on factors such as run-off conditions and the progression of the underlying atherosclerosis.

There are several other options for revascularisation after the removal of an infected vascular graft in the groin, including semi-closed endarterectomy (ring-stripping) or balloon angioplasty of the native iliac artery, axillofemoral bypass by lateral route avoiding the infected groin [28, 29], and subscrotal bypass [30]. However, the OFB gives better results than bypass through these alternative extra-anatomical routes. If the groin is not grossly infected, then an autologous bypass of saphenous [31] or femoral vein [32] or thrombectomised femoral or iliac artery may be placed in situ without causing major problems, although the danger of future graft rupture is always present [33]. In addition, in situ revascularisation with a rifampicin-impregnated graft may give satisfactory results [34]. **[Q8: A, B, C, E, F]**

Although the obturator bypass procedure is not used frequently, it should be a part of the vascular surgeon's armamentarium. It may be effective in solving a difficult revascularisation problem in the groin, if performed appropriately.

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23. Diabetic Foot

Mauri J. A. Lepäntalo, Milla Kallio and Anders Albäck

A 54-year-old smoker with type 2 diabetes of 7 years duration had a minor abrasion to the lateral aspect of the left fifth toe. The patient was known to have hypertension, nephropathy and retinopathy, and he was overweight. His glycaemic control was good following recent addition of insulin to his oral medication. The superficial ulcer did not bother the patient, and it was initially followed up in his local healthcare centre. Two months later, the patient was referred to a community hospital because of infection and suspicion of osteomyelitis. He now had an infected ulcer lateral to the head of the fifth metatarsal, with a discharge. Plain X-ray films showed suspected osteomyelitis. Dorsalis pedis and posterior tibial pulses were reported to be present. The C-reactive protein (CRP) level was 31 mg/l, leucocytes 14.8×10^9/l, and blood glucose 12 mmol/l.

Question 1

What condition(s) are likely to be responsible for the foot problem?

A. Infection.

B. Atherosclerotic macroangiopathy.

C. Diabetic microangiopathy.

D. Neuropathy.

Question 2

What is the simplest tool available in the surgery or outpatient clinic to detect osteomyelitis?

A. Plain X-ray films.

B. Clinical examination with blunt nasal probe.

C. Magnetic resonance imaging.

D. Computer tomography.

Question 3

What simple tools are available in the surgical outpatient clinic to assess angiopathy?

A. Palpation of foot arterial pulses.

B. Examination of audible signal with hand-held continuous wave Doppler.

C. Ankle pressure measurement.

D. Duplex scanning of lower extremity arteries.

Question 4

What simple tools are available in the surgery or outpatient clinic to assess neuropathy?

A. Monofilament sensation testing.

B. Achilles tendon reflex.

C. Tuning fork testing.

D. Electroneuromyography (ENMG).

The patient was admitted to the medical ward for treatment of his infected foot. Despite the administration of intravenous antibiotic treatment, later modified according to the results of bacterial cultures, the infection progressed. One week after admission, lateral and superficial plantar compartments were drained operatively on the lateral side of the fifth metatarsal head and between the fourth and fifth metatarsal heads. Abundant pus was obtained, and the fifth metatarsal head was observed to be soft. The operative wound was left open. The infection seemed to subside, and the patient was discharged after a 16-day admission with oral clindamycin treatment and local wound care.

Question 5

What major problems were neglected at this point?

A. Presence of osteomyelitis.

B. Presence of ischaemia.

C. The wound was left without coverage with split thickness skin grafting.

D. The weight-bearing wound area of the foot was not protected with a cast.



Fig. 23.1. Foot at the time of admission to the vascular unit.

Despite continuous antibiotic treatment and local treatment of the open lesion on the lateral aspect of the foot, the situation worsened over the next 2 months and the patient was readmitted to the hospital. The patient had fever and his CRP level was 123 mg/l. The serum creatinine was 1.6 mg/dl. An immediate wound debridement and amputation of the fourth toe was performed, after which the patient was admitted to a vascular surgical unit (Fig. 23.1). There was a faint popliteal pulse with no other pulses palpated distally. Ankle brachial indices (ABIs) were 1.35 and 1.21. The patient could not feel the touch of the monofilament on the plantar surface of the great toe or the first and fifth metatarsal heads.

Question 6

How would you further examine the circulation non-invasively or invasively?

A. Toe pressure measurement.

B. Ankle pressure measurements and pulse wave recordings.

C. Treadmill test with pressure measurements.

D. Duplex scanning of distal arteries.

E. Magnetic resonance angiography.

F. Digital subtraction angiography.

The toe pressures were 73 mm Hg on the right side and 29 mm Hg on the left side. A selective angiography was obtained the next day (Fig. 23.2).

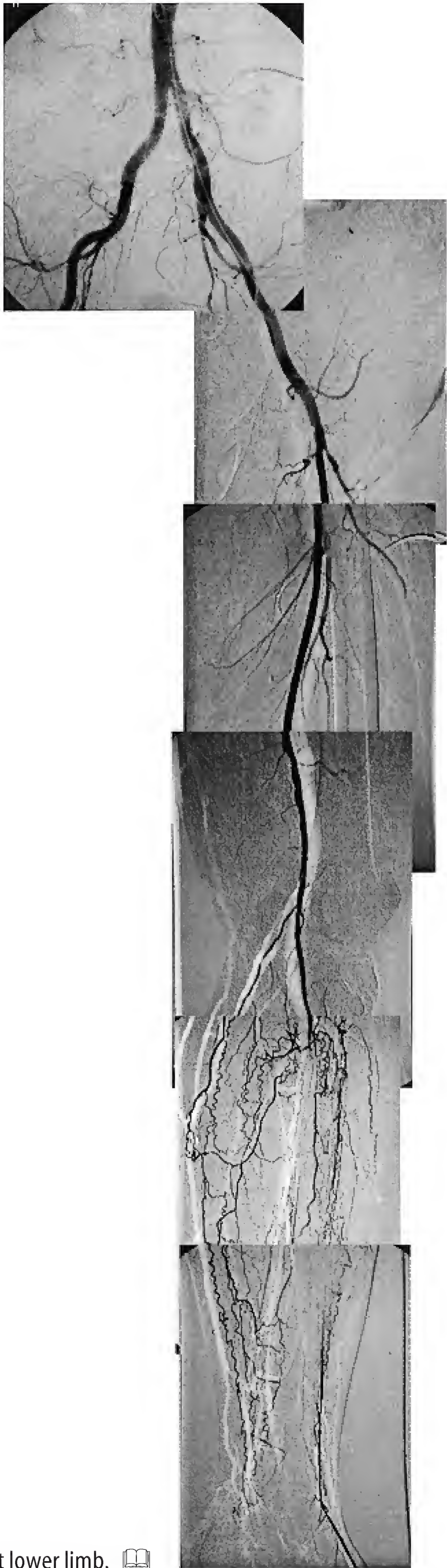


Fig. 23.2. Angiography of the left lower limb. 🕮

Question 7

What angiographic findings typical of diabetes can you see?

A. Normal aortoiliac segments.

B. Haemodynamically non-significant occlusive disease of crural vessels.

C. Significant occlusive disease of crural vessels.

D. Severe occlusive disease of all foot vessels.

E. Patent foot vessel.

Question 8

What treatment strategy would you prefer?

A. No possibilities for reconstruction. Choose the best medical treatment, then wait and see.

B. No possibilities for reconstruction. Foot-level amputation up to bleeding tissue.

C. Below-knee amputation.

D. Possible acute debridement, reconstruction to pedal artery, and further wound excision later.

E. No wound excision and reconstruction to pedal artery until the wounds are clean.

Question 9

If you consider vascular reconstruction, what would be your preferred inflow site in this patient?

A. Common femoral artery.

B. Superficial femoral artery.

C. Popliteal artery.

A popliteopedal reconstruction was made 5 days after admission to the vascular surgical unit. The great saphenous vein was used in situ with the supragenicular popliteal artery as a recipient vessel. Despite achieving an acceptable initial flow of 51 ml/min, the graft thrombosed the next day and a thrombectomy and a revision of the graft was made. A narrow segment below the knee was replaced with a reversed proximal great saphenous vein under angioscopic control. A flow of 110 ml/min was measured with transit time flowmetry.

Question 10

Which of the following methods are adequate for intraoperative control?

A. Angiography alone.

B. Doppler alone.

C. Flowmetry alone.

D. Flowmetry with a method giving morphological information.

E. Intraoperative duplex scanning alone.

The postoperative ABI was 0.97. Wound excision and three-ray amputation of the lateral toes were performed 2 days after revascularisation. The patient was discharged 2 weeks after admission and transferred to the community hospital. Split thickness skin grafting was performed there. The patient was discharged home with a heel-sandal (a sandal in which the body weight is borne only by the heel), antibiotic treatment for one more week, and local wound care. The healing of the wound progressed well. Six weeks after the vascular reconstruction, the patient was pre-



Fig. 23.3. Foot at 1-year follow-up.

scribed insoles. He also used a silicon piece correcting the position of the second toe (Fig. 23.3).

At 1-year follow-up, ABI was greater than 1.3/0.91 and toe pressures were 65/55 mm Hg. Duplex surveillance findings indicated a possible vein graft stenosis.

Question 11

What are the findings indicating vein graft stenosis in the duplex examination?

A. Midgraft peak systolic velocity (PSV) of less than 45 cm/s.

B. V2/V1 ratio greater than 3 (V2, PSV at the site of the maximum stenosis; V1, PSV in the normal graft adjacent to the stenosis).

C. Maximum PSV greater than 300 cm/s.

D. End-diastolic flow velocity (EDV) greater than 20 cm/s.

A control angiography was performed, but no severe stenosis was found (Fig. 23.4).

Commentary

This case illustrates the problems related to delayed diagnosis and treatment of diabetic neuroischaemic foot. The aetiology of diabetic foot ulceration and infection is multifactorial. Our patient evidently had infection and probably also neuropathy. Neuropathy often abolishes sensation, and an unpleasant odour and discharge may be the first signs of infection to the patient, especially if the lesion is situated on the plantar aspect of the foot. The role of microangiopathy in diabetic foot is not confirmed, but ischaemia due to atherothrombotic disease often plays a major role [1]. **[Q1: A, B, D]**

The simplest method is to examine the ulcer with a blunt nasal probe. If it hits the bone, then osteomyelitis is most likely. The diabetic wounds should be classified systematically according to a precise system, such as the Armstrong classification (Table 23.1), which takes into account both the depth of the lesions and the presence of ischaemia and infection [2]. Plain X-ray films are of limited value and magnetic resonance imaging (MRI) is the most reliable tool for diagnosis of osteomyelitis [3]. **[Q2: B]**

The patient was reported to have palpable distal pulses at one time but not at another time. Furthermore, the popliteal pulse was reported to be palpable and ABI to be normal. Palpation of foot pulses is not a fully reproducible observation, and they may be considered normal if both tibialis posterior and dorsalis pedis pulses are clearly felt [4]. If either is not palpated, non-invasive evaluation is necessary. It is far more difficult to palpate the popliteal pulse, and it has been suggested that if an inexperienced palpator feels the popliteal pulse, this indicates an aneurysm. Systolic pressure measurements taken at the level of the ankle by a Doppler device are the most common non-invasive method for assessment of atherothrombotic disease. However, the results may be biased due to the presence of mediasclerosis, which is present in 15–40% of diabetics [5]. Incompressible arteries may allow the



Fig. 23.4. Control angiography after 1-year follow-up. 📖

Table 23.1. Classification of diabetic foot lesions by grading and staging according to the depth of the lesion and the presence of infection and ischaemia, as proposed by Armstrong et al. [2]. Updated

Depth	
	Grade 0: pre- or post-ulcerative site which has healed
	Grade I: superficial wound through the epidermis or epidermis and dermis which does not penetrate to tendon, capsule or bone
	Grade II: wound which penetrates to tendon or capsule
	Grade III: wound which penetrates to bone or joint
Infection and ischaemia:	
	Stage A: clean wound
	Stage B: non-ischaemic infected wound
	Stage C: ischaemic non-infected wound
	Stage D: ischaemic infected wound to tendon, capsule or bone

signal to be heard in cuff pressures as high as the patient tolerates. In patients with mediasclerosis, the ABI typically exceeds 1.15 [6]. The audible Doppler signals may help the examiner, as an open inflow channel gives high-pitched biphasic signals but collateral flow around an occlusion usually gives only a low-pitched monophasic murmur. **[Q3: A, B, C]**

Symptoms of neuropathy include loss of sensation, hyperaesthesia and burning, and aching pain, which are often worse at night [7]. Many patients with severe neuropathy are asymptomatic. Achilles tendon reflex, monofilament sensation testing and 128-Hz tuning fork testing are other recommended clinical tests [8]. **[Q4: A, B, C]**

The primary diagnostic work-up in this case was clearly deficient. The patient obviously had osteomyelitis, which would have necessitated prompt drainage and amputation. Furthermore, the role of ischaemia should also have been evaluated and corrected within 3–5 days after proper drainage. **[Q5: A, B]**

The Doppler-derived pressures were clearly pseudohypertensive due to the arterial wall stiffness. Pseudohypertension affects digital arteries far less frequently, and therefore toe pressures are more reliable. A pulse volume recording at the ankle can also help to detect mediasclerosis. Another method is to measure systolic blood pressure at the ankle with Doppler but without an occluding cuff – the pole test [9]. The examiner listens to the Doppler signals of the supine patient while the foot is elevated gradually until the signals disappear. The scale in the pole gives the pressure at the ankle (0.75 × pressure (cm) equals the pressure (mm Hg)). In centres where duplex scanning of distal arteries can be done by trained validated examiners this method would preferably be the next investigation. Magnetic resonance angiography is also a method of choice if high-quality images are available and especially when the patient suffers from marked nephropathy. On the other hand in centres where technically demanding endovascular procedures can be done during diagnostic contrast angiography this is – as in our case – the primary imaging technique. **[Q6: A, B, D, E, F]**

Atherosclerotic changes in diabetes are typically situated in femoral and crural arteries, or only in crural arteries, in contrast to non-diabetic patients who tend to have the first symptoms from the obliteration of the aortic bifurcation. Despite proximal crural artery occlusion, the pedal arteries may be patent, as was the dorsalis pedis artery in this patient. **[Q7: A, C, E]**

The treatment strategy is affected strongly by the presence and severity of infection. A superficial ulceration may be only the tip of the iceberg. There may be

penetration, hidden to the eye, into deep tissues. Vigorous debridement must be carried out to establish the degree of penetration and to remove all necrotic tissue [3]. Fulminant infection may necessitate guillotine amputation. The bypass can often be performed 3–5 days after debridement. If ischaemia plays a major role and the infection is quiescent, then revascularisation can, in selected cases, be performed first. Vascular reconstruction can be performed in as many as 90 per cent of diabetic patients with atherothrombotic disease [10]. The best outflow vessel in continuity with the foot should be selected [11]. Diabetes is not considered to affect the outcome of graft patency, although female diabetic patients are reported to have worse outcome regarding patency and leg salvage [12]. In limbs with large tissue defects, a microvascular free muscle flap transfer can be used for defect coverage in conjunction with long bypass [13]. **[Q8: D]**

Short bypasses do well if the inflow artery is not compromised, as in our patient. Although the above-knee popliteal artery gave better results as the inflow vessel than the below-knee popliteal artery in our own series [14], the question is not settled. **[Q9: C]**

Angiography is the gold standard for intraoperative monitoring. The accuracy of flowmetry is affected strongly by the reproducibility of the method. In contrast to older methods, transit-time flowmetry, which does not require information on the diameter of the vessels, has proven to be very accurate [15]. Despite this, it gives only flow values and does not inform about the morphology. The present case clearly shows how the typically narrow segment of the great saphenous vein below the knee was missed despite good flow during the initial hyperaemia. In this area, there was an intimal tear caused by the valvulectomy catheter. Unfortunately, an angioscope was not used in the first operation. An angioscope visualises the inner surface of the vessel, whereas intravascular ultrasound is better for detecting changes within the vessel wall. Doppler and duplex may be used for intraoperative monitoring as well. Doppler gives only haemodynamic information, whereas duplex gives a combination of anatomical and haemodynamic information. There is no best method for intraoperative monitoring, but the optimal method would be to have both haemodynamic and morphological information. **[Q10: A, D, E]**

As there is a 30 per cent risk of developing neointimal hyperplasia and graft stenosis within the first postoperative year, duplex surveillance is considered an essential part of postoperative care. All the suggested duplex criteria are indicative of vein graft stenosis, but none of them can be 100 per cent sensitive in detecting stenosis [16]. Our case demonstrates that using liberal duplex criteria, false positive findings are easily encountered as the angiography was deemed normal. **[Q11: A, B, C, D]**

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24. Chronic Visceral Ischaemia

George Geroulakos

A 68-year-old woman presented with a 19-month history of generalised abdominal pain. Initially, she experienced the pain following meals, but subsequently the pain became persistent. Over this period of time, she lost 12 kg in weight. For the last few months before admission, she started having diarrhoea once to twice per day. There was no blood or mucus in the stool. Her past medical history included partial gastrectomy 17 years earlier for benign disease.

On examination, the patient looked cachectic. Her abdomen was slightly distended, and the bowel sounds were increased. There was a high-pitched epigastric bruit. Routine blood tests were normal.

Question 1

Which is the likely diagnosis for our patient on the basis of the available information so far?

A. Cancer of the pancreas.

B. Peptic ulcer.

C. Subacute intestinal obstruction secondary to adhesions.

D. Mesenteric angina.

E. Cancer of the large bowel.

Faecal fat measurement was 17.6 g/day (normal value <6 g/day). Gastroscopy was performed, which showed features compatible with atrophic gastritis. This was followed by computed tomography (CT) scanning of the abdomen, which reported that the pancreas could not be defined well as a result of paucity of retroperitoneal fat. In addition, CT showed non-specific thickening of the small-bowel loops. Endoscopic retrograde cholecystopangreatography (ERCP) was performed, which ruled out pancreatic pathology. A small-bowel enema did not demonstrate any significant findings. A colonoscopy was performed, which showed two isolated

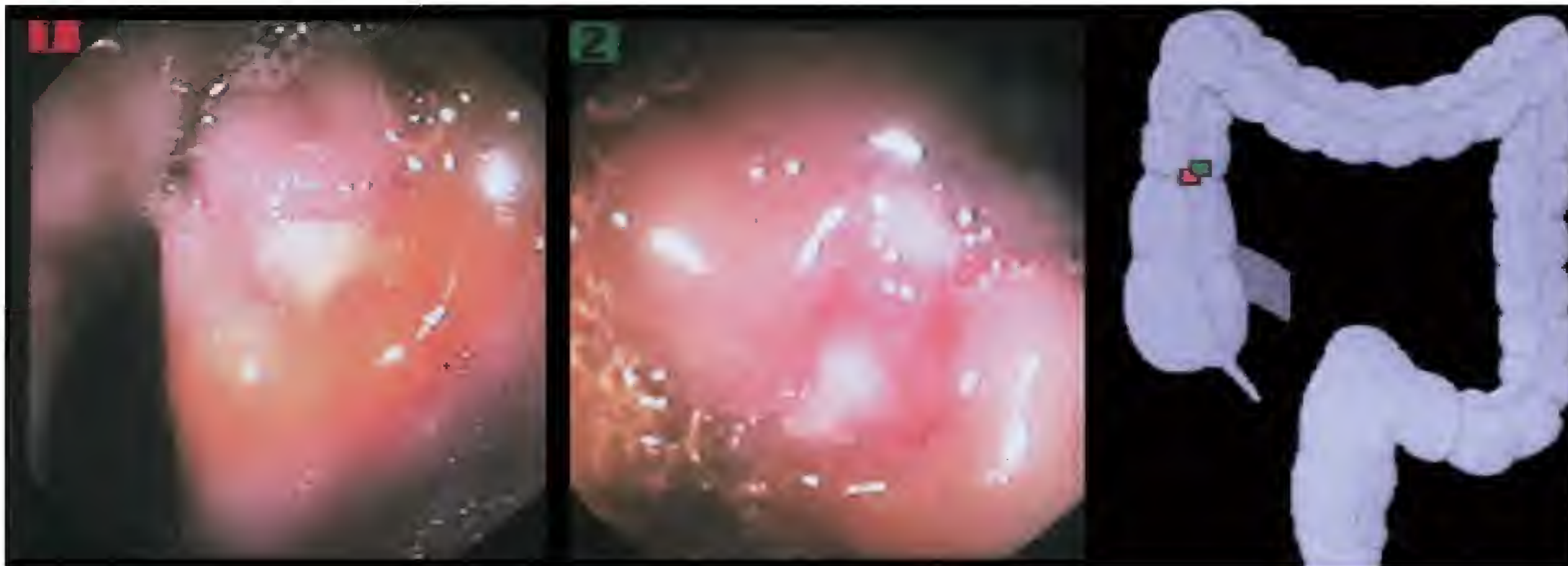


Fig. 24.1. Colonoscopic view of an isolated ulcer in the ascending colon in a patient with chronic visceral ischaemia.

ulcers in the ascending colon (Fig. 24.1) and raised the possibility of ischaemic colitis. Fig. 24.2 shows the lateral aortogram of our patient, which showed occlusion of the coeliac artery and 95 per cent stenosis of the superior mesenteric artery. A diagnosis of chronic visceral ischaemia was made.



Fig. 24.2. Lateral aortogram demonstrating occlusion of the coeliac artery and a 95 per cent stenosis of the superior mesenteric artery.

Question 2

Which of the following statements regarding chronic visceral ischaemia is correct?

A. It has a marked male preponderance.

B. As described in our patient, it usually takes more than 1 year from the first presentation of the symptoms until the final diagnosis is made.

C. It presents clinically as an undiagnosed malignancy.

D. Symptoms occur when at least one of the three visceral arteries has significant disease.

E. It may cause malabsorption.

The patient underwent antegrade revascularisation via a ninth rib extraperitoneal thoracoabdominal approach of the coeliac artery and the superior mesenteric artery. An 8-mm Dacron graft was used as a conduit.

Question 3

Which of the following statements regarding the management of this patient are false?

A. The best patency can be achieved using a venous conduit.

B. Revascularisation of the coeliac artery was unnecessary, and equally good results could have been achieved with revascularisation of only the superior mesenteric artery.

C. Surgical revascularisation should not have been considered in this elderly, frail patient because it has an excessive mortality rate of about 30 per cent in most series.

D. Percutaneous transluminal angioplasty (PTA) should have been the method of choice.

The postoperative recovery of the patient was uneventful. She was discharged home on the eighth postoperative day. Six months later, she was asymptomatic and had gained 5 kg in weight. However, at 12 months the patient presented to the outpatient clinic with recurrent postprandial abdominal pain. A duplex examination showed that the graft to superior mesenteric artery anastomosis had more than 60 per cent stenosis and the graft to coeliac artery anastomosis was occluded.

Question 4

What would you advise your patient?

A. Reoperation aiming to revascularise the coeliac artery and place a patch on the graft to superior mesenteric artery anastomosis.

B. Angioplasty and stenting of the graft to superior mesenteric artery anastomosis.

C. Conservative management advising the patient to take small and frequent meals.

The patient underwent angioplasty and stenting of the graft to superior mesenteric artery anastomosis with an excellent technical and clinical result. Twenty-four months following this procedure the patient remains asymptomatic.

Commentary

As described in our patient, the clinical picture of chronic visceral ischaemia includes abdominal pain with or without diarrhoea and weight loss. The diagnosis of chronic visceral ischaemia is in doubt if the patient has no significant decrease in total body mass. The abdominal pain occasionally radiates to the back. The pain of visceral ischaemia has similarities to that of carcinoma of the stomach, pancreatic carcinoma and peptic ulceration. Diarrhoea may be explained by the increased motility of the bowel induced by the ischaemia; it may also be secondary to malabsorption. **[Q1: A, B, D, E]**

Other symptoms that may be seen include nausea and vomiting, which have been associated with gastric motility disorders caused by ischaemia [1]. An epigastric bruit may or may not be present. Our group and others have reported a marked female patient distribution of this condition [2–4]. The reason for this peculiar sex distribution remains undetermined. However, it has been suggested that it could be the result of the inclusion of cases of Takayasu's aortitis in reports of atherosclerotic chronic visceral ischaemia [5]. Takayasu's aortitis closely mimics atherosclerosis of the abdominal aorta and has a marked female predominance.

The time from the onset of symptoms to diagnosis is usually more than 12 months [6]. The diagnosis of chronic visceral ischaemia is a clinical one. As shown clearly in our case, contrast studies, abdominal ultrasound, endoscopy and CT are not essential to the diagnosis but will prove important in eliminating other sources of abdominal discomfort. In all instances, lateral views of biplane aortography demonstrate visceral occlusive lesions compatible with the diagnosis. As a result of an abundant network of collateral vessels, clinical symptoms are present when at least two of the three visceral arteries have significant disease. There are known asymptomatic cases with all three visceral arteries thrombosed, thus emphasising the fact that chronic visceral ischaemia cannot be diagnosed exclusively on the basis of X-rays. **[Q2: B, C, E]**

Techniques of revascularisation include transection and reimplantation, bypass grafting, endarterectomy and balloon angioplasty with or without stent placement. There is no consensus regarding the best surgical approach for the treatment of chronic visceral ischaemia. This condition is encountered infrequently, and it is unlikely that a single centre can treat enough patients and accumulate sufficient experience to develop principles of treatment by demonstrating significant differences between the various mesenteric revascularisation strategies. Bypass grafting is the most common type of visceral revascularisation performed; it may originate from several different locations, including the supracoeliac aorta, the infrarenal

aorta and the common iliac arteries. Regardless of the bypass technique used, the status of the donor artery is critical to success [7]. The distal thoracic aorta is usually free of atherosclerotic disease and is an excellent origin of a short antegrade bypass to the superior mesenteric artery. The bypass is placed in the direction of normal blood flow, thus reducing anastomotic turbulence. In addition, this design eliminates the possibility of kinking and thrombosis by compression or traction from the overlying intestinal mesentery, which may be observed with retrograde grafts originating from the infrarenal aorta or the iliac arteries. The distal portion of the thoracic aorta may be approached from the abdomen through division of the crura.

There is no uniform agreement about the graft material of choice. In early reports, vein grafts had patency rates inferior to synthetic grafts [9, 10]. More recent reports described the use of either autogenous veins or prosthetic grafts with excellent long-term function and no difference in patency rates [11, 12]. In our case, we used synthetic Dacron bypass because it is always available, spares the patient from the morbidity of one or more incisions for the harvesting of the vein, and provides good early and long-term results.

Aorto-superior mesenteric artery bypass alone is usually sufficient to provide good symptomatic relief as a result of the extensive collateral circulation, even when all three visceral arteries are occluded. Hollier et al. [13] have shown that complete revascularisation in multivessel disease resulted in a late recurrence of 11 per cent, while when one of three stenotic vessels was revascularised the recurrence rate was 50 per cent. They concluded that it is preferable to revascularise as many vessels as possible to provide the best chance of long-term relief.

Most recent series report an acceptable operative mortality rate ranging from 3 to 8 per cent.

Our patient could have been considered for angioplasty of the superior mesenteric artery. There is limited information about the selection of patients for PTA and the long-term results. A number of small series reported early technical success in 70–100 per cent of patients. Recurrence after PTA is greater than after surgery. In a series of 18 patients with chronic visceral ischaemia who had a technically successful angioplasty, symptomatic recurrence occurred in three (20 per cent) patients at a mean interval of 28 months, while three patients had only partial symptomatic relief [14]. Angioplasty is safer than surgery, although fatal distal embolisation and fatal superior mesenteric artery dissection with thrombosis and bowel infarction have been reported. At present, it should probably be limited to high-risk patients. PTA of the visceral arteries can be difficult when performed via the femoral artery approach. Access through the brachial and axillary artery may be easier, but this route does not guarantee technical success. **[Q3: False A, B, C, D]**

Recurrent visceral ischaemia is not uncommon after primary visceral revascularisation for chronic visceral ischaemia. In a large series of 109 patients who underwent primary visceral revascularisation at the University of California, San Francisco over a period of 38 years, 19 patients had recurrent visceral ischaemia, 12 (11 per cent) patients had recurrent chronic visceral ischaemia, and seven (6.4 per cent) had acute visceral ischaemia [15]. The minimally invasive nature of the endovascular techniques and the increased complication rate of reoperations renders the endovascular approach a reasonable first option in properly selected patients with recurrent symptoms [16]. **[Q4: B]**

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25. Acute Mesenteric Ischaemia

Jonathan S. Refson and John H. N. Wolfe

A 78-year-old woman presented to the emergency department with a 12-h history of sudden-onset abdominal pain. She had vomited after the pain started, and she had also had two episodes of diarrhoea. Until this time, she had been well, although she was known to be in atrial fibrillation and took digoxin 125 mg daily.

On examination, she was distressed and obviously in pain. Baseline observations revealed a pulse of 110 bpm, irregularly irregular, blood pressure of 95/60 mm Hg, respiratory rate of 28 breaths/min, and temperature of 37.3°C. Her chest was clear, heart sounds were normal (irregular rhythm), and the jugular venous pressure was not elevated. Abdominal examination was unremarkable, with a soft abdomen and minimal tenderness despite severe pain, and normal bowel sounds.

The investigations shown in Table 25.1 were performed by the admitting surgeon.

Electrocardiogram (ECG) revealed atrial fibrillation with no other acute changes. Erect chest X-ray revealed normal lung fields and no free gas under the diaphragm. Abdominal radiography was unremarkable except for minimal small-bowel distension.

Question 1

Which of the following is the most unlikely diagnosis?

A. Acute ulcerative colitis.

B. Pancreatitis.

C. Mesenteric venous thrombosis.

D. Acute mesenteric ischaemia (AMI).

E. Diabetic ketoacidosis.

Table 25.1. Investigations performed by the admitting surgeon. Updated 🕮

Investigation	Finding
Urinalysis	No abnormality
Biochemistry	Na^+ 139 mmol/l K^+ 4.6 mmol/l Creatinine 112 mmol/l Glucose 6.1 mmol/l Amylase 2000 IU/l
Haematology	Haemoglobin 12.3 g/dl White cell count 27,000 Platelets 235,000
Arterial blood gas	pH 7.21 pCO_2 3.2 kPa pO_2 9.4 kPa HCO_3^- 17 mmol/l Base excess –8

Question 2

What are the most common causes of AMI?

A. Renal failure.

B. Atrial fibrillation.

C. Multi-organ failure.

D. Anti-phospholipid syndrome.

E. Atherosclerotic disease

Question 3

Which of the following tests are of use in the acute management of a patient with AMI?

A. Echocardiography.

B. Lateral-view mesenteric angiography.

C. Thyroid function tests (TFTs).

D. Non-contrast computed tomography (CT) scanning.

E. Mesenteric vessel duplex Doppler.

At this point, the patient was taken to the high-dependency unit, where the following measures were undertaken: high-flow oxygen therapy by mask (15 l/min), continuous ECG monitoring, central venous pressure (CVP) monitoring, urinary catheter inserted to monitor urinary flow hourly, and infusion of 4 litres of fluid resuscitation. Intravenous broad-spectrum antibiotics and an anticoagulant dose of

Table 25.2. Repeat blood gas and blood count investigations. Updated

Investigation	Finding
Haematology	Haemoglobin 10.2 g/dl White cell count 37,000 × 10^9/l Platelets 235 × 10^9/l
Arterial blood gas	pH 7.19 pCO_2 3.1 kPa pO_2 49.4 kPa HCO_3^- 11 mmol/l Base excess −15

intravenous heparin were also given. After 2 h of resuscitation, the patient's blood pressure was 130/85 mm Hg, pulse 100 bpm and CVP +8 cm water. She was still in a lot of pain despite 10 mg of diamorphine, and she was still tachypnoeic. Repeat blood gas and blood count investigations were as in Table 25.2.

Because the patient was persistently acidotic with an elevated white count and in severe pain, she was taken to the operating theatre for an emergency laparotomy. Almost the entire small bowel and most of the large bowel were found to be ischaemic but viable. There was a pulse in the proximal superior mesenteric artery (SMA) but nothing was palpable beyond the origin of the middle colic vessel.

Question 4

What operative options are available to achieve restoration of flow to the bowel?

A. Full heparinisation.

B. Catheter thrombectomy.

C. Axillofemoral bypass.

D. Mesenteric bypass with a vein graft.

E. Mesenteric bypass with prosthetic graft.

Clot was removed successfully from the SMA. However, despite the majority of the bowel receiving a good blood supply, several areas remained dusky in appearance.

Question 5

What features of the bowel's appearance determine whether it is viable?

A. The presence of peristalsis.

B. Lack of foul odour from the peritoneal cavity.

C. Serosal sheen.

D. Mesenteric pulsation.

E. Active bleeding from the cut surface of the bowel at the time of resection.

Question 6

Having determined that an area of the bowel is non-viable, what action should you take?

A. Revascularise the bowel, then remove that which is non-viable.

B. Remove the non-viable bowel, then revascularise the remaining bowel.

C. Resect all non-viable bowel and primarily anastomose ends; then close the abdomen.

D. Close the abdomen and start the patient on an intravenous infusion of diamorphine.

E. Resect all non-viable bowel and exteriorise viable ends; plan relook laparotomy.

Commentary

The incidence of AMI is approximately 1/100,000 in the UK and USA [1, 2]. AMI is a life-threatening vascular emergency. It accounts for 0.1 per cent of emergency admissions [3], and based on several large series it has a mortality of between 60 and 100 per cent [3–7]. Females are affected twice as often as males, and the median age at presentation is 70 years [6].

The clinical presentation is often not as clear-cut as described in the case above. However, some if not all of the described features will be present. One must have a high index of clinical suspicion in anyone aged over 55 years who presents with abdominal pain out of proportion to the physical signs elicited on abdominal examination [8]. The diagnosis should also be considered in patients with known peripheral arterial disease and abdominal pain. The triad of abdominal pain, a cardiac source of embolus and gut emptying, as described by Klass [9], make AMI the most likely diagnosis. In addition to this triad, the finding of a marked leucocytosis, metabolic acidosis and hyperamylasaemia are also suggestive of AMI. It is also not unusual for there to have been a history of previous embolic events [8]. Pancreatitis can be difficult to differentiate from AMI, and laparotomy is indicated if suspicion of AMI is aroused. **[Q1: E]**

Defining the aetiology of AMI is important as the different causes have different treatments. The most common presentation, as described in our case, is of superior mesenteric embolus; this accounts for about 50 per cent of all cases [3, 5–7]. The usual source for these clots is the atria in patients in atrial fibrillation or the ventricle if the patient has recently sustained a myocardial infarction. Another potential source of emboli is atheroma from the aortic wall following radiological procedures in which catheters and guidewires have been passed up the aorta. The consequence of such an event can be catastrophic, as the mesenteric circulation has not had time to develop a collateral circulation.

The next most frequent aetiology is SMA thrombosis, which accounts for between 25 and 50 per cent of cases [3, 5-7]. This results from progression of atheromatous disease at the origin of the SMA. It is important to note that a long-standing stenosis may have caused symptoms of chronic mesenteric ischaemia in the months before its ultimate occlusion [10]. Therefore, in a patient with pre-existing symp-

toms of mesenteric ischaemia, sudden onset of abdominal pain should be regarded as AMI until proven otherwise.

Non-occlusive mesenteric ischaemia (NOMI), first described by Ende in 1958 [11], is the next most frequently encountered condition, occurring in about 20 per cent of cases [3, 5, 6]. In this situation, the patient is often critically ill from another cause and the mesenteric ischaemia is due to vasoconstriction leading to reduced flow in the splanchnic circulation. This may be due to cardiogenic shock, hypovolaemia or vasoconstricting inotropes. Even after reversal of shock, mesenteric hypoperfusion may persist for several hours [12, 13].

Mesenteric venous thrombosis (MVT) is the least common cause and accounts for about 5 per cent of AMI [14–16]. The thrombotic process is thought to start in the superior mesenteric vein and spreads to the portal vein; the inferior mesenteric vein is usually spared. The onset of symptoms is more insidious and may have a history of several days. It is caused by the same provoking factors that one finds in any thrombotic situation: sluggish flow, clotting abnormality and vessel wall damage (Virchow's triad). It is associated most commonly with hypercoagulable states, abdominal trauma or intra-abdominal sepsis [17–20]. The diagnosis is often made at laparotomy and encompasses a spectrum of severity from segmental mesenteric venous thrombosis to the entire portal vein being thrombosed. **[Q2: B, E]**

Diagnostic confirmation of AMI poses a dilemma. Should one delay in order to confirm a suspicion and risk converting a salvageable situation into a non-salvageable one [21]? There is little evidence on which to base sound advice. However, if the patient is cardiovascularly stable with minimal symptoms, and one has prompt access to angiography, then this provides accurate diagnosis (Fig. 25.1). Some authors recommend colour-flow duplex at the bedside during the resuscitation phase [22, 23]; this procedure is less time-consuming than angiography, but it requires considerable skill that is not always available. Furthermore, good views are often hampered by obesity and/or overlying bowel gas.

Transthoracic echocardiography is useful in identifying a cardiac source for emboli and may help in making the decision regarding postoperative anticoagulation. However, it is not as sensitive as transoesophageal echo in searching for left atrial emboli and may waste valuable time.

Contrast-enhanced CT may be of use in identifying mesenteric venous thrombosis [24]. This will not prevent laparotomy as bowel resection may well be necessary. If there is clear evidence of peritonism and a high index of suspicion for AMI, then the patient should be resuscitated rapidly and this should be followed by urgent laparotomy. **[Q3: B, E]**

In order to answer Question 4, one has to be confident of the aetiology of AMI. In our case, there is embolus in the SMA. The abdomen should be approached through a long midline incision, which will afford excellent exposure. Having entered the abdomen, a quick survey of the viscera and extent of ischaemia should give some information on the aetiology of the AMI (see below). At this point, the main aim of surgery is to restore flow to the ischaemic viscera if viable. In order to do this, the transverse mesocolon is elevated and the ligament of Treitz identified and the fourth part of the duodenum mobilised. The root of the mesentery is palpated to feel for the SMA pulse. If, as in our case, the AMI is due to an embolic event, then a proximal SMA pulse should be palpable and the duodenojejunal flexure and proximal few centimetres of jejunum should be viable. The emboli usually lodge at a variable site 3–8 cm

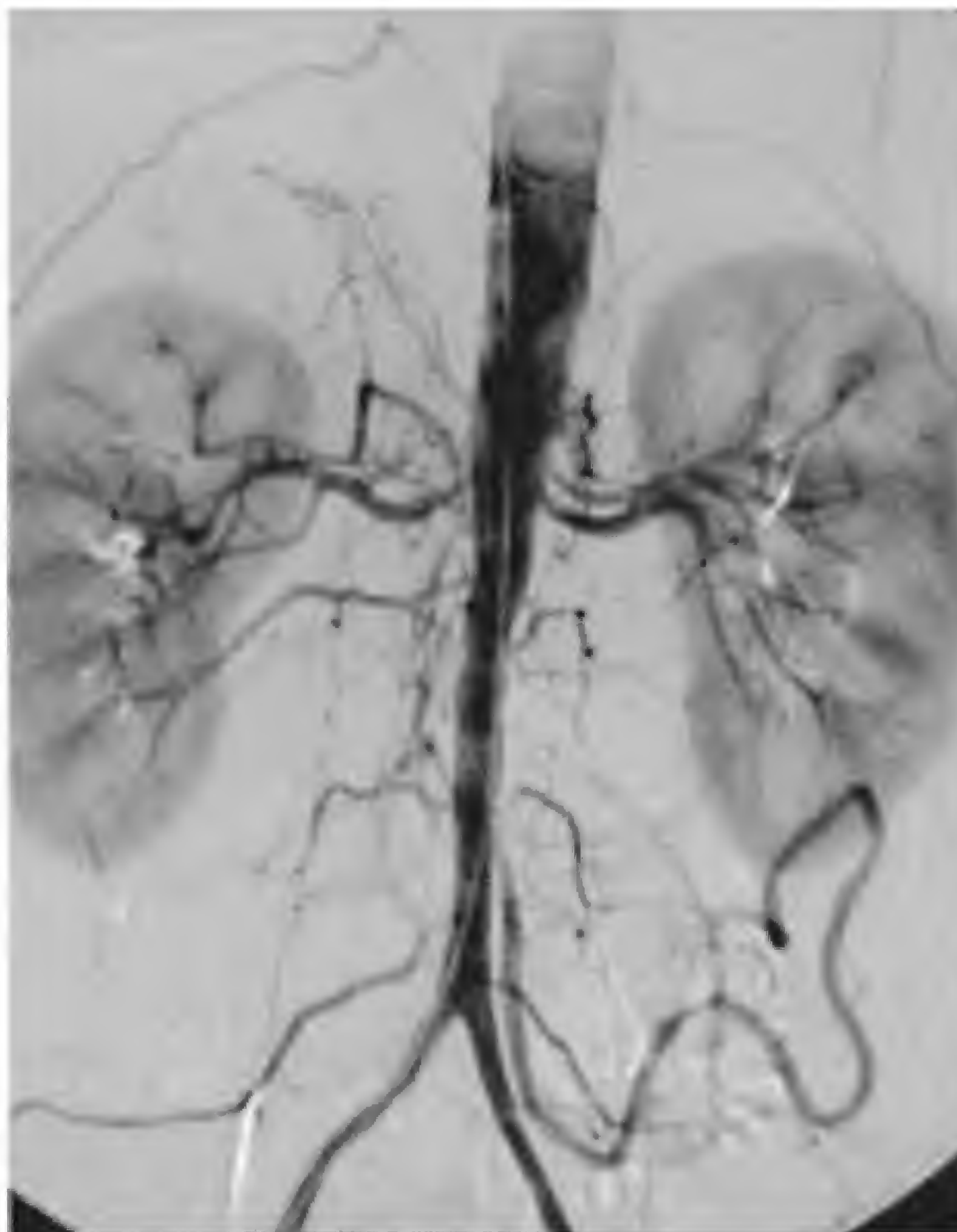


Fig. 25.1. Narrowed atherosclerotic aorta with no coeliac or superior mesenteric filling.

from the origin of the SMA, usually at the point where the middle colic artery arises [6, 8, 21] (Fig. 25.2a, b).

In order to expose the SMA, the inferior leaflet of the mesocolon is entered along the course of the vessel and a 5-cm section of artery is dissected out and slung with Silastic sloops. At this point, if the patient has not been heparinised previously, then they should be given an intravenous dose of 5000 units of unfractionated heparin. A longitudinal arteriotomy is made in the cleared SMA (on the left margin of the vessel, to permit easier graft placement, should a bypass be necessary) [21], and a size 3 or 4 Fogarty embolectomy catheter is passed up and down the vessel to retrieve the embolus. Once adequate backward and forward flow has been achieved, the vessel should be flushed with heparinised saline; the arteriotomy can then be closed primarily or with a patch (depending on size), using a 6/0 or 7/0 Prolene suture.

At this point, the anaesthetist should be warned that you are about to reperfuse the viscera. Metabolites that have accumulated in the ischaemic viscera will pass rapidly from the mesenteric venous circulation into the systemic circulation, which can precipitate circulatory collapse and, over several hours, result in the development of systemic inflammatory response syndrome (SIRS). There is no point in revascularising dead bowel; indeed, this is dangerous. Irrefutably dead bowel should be removed before revascularisation. It is important to note that if, on



a



b

Fig. 25.2. a, b Patchy mid-gut infarction due to atherosclerotic occlusion of superior mesenteric artery.

opening the abdomen, the entire small bowel is black and irreversibly ischaemic, then the most appropriate thing to do is close the abdomen and keep the patient comfortable with morphine: death will usually follow within a matter of hours.

In the event that there is no pulse palpable at the SMA origin, then the revascularisation strategies are similar to those for chronic visceral ischaemia (see Chapter 24). In this case, attempts at thrombectomy will fail as the catheter will not cross the occlusion. The options available are retrograde bypass from the infrarenal aorta to the SMA, antegrade bypass from the supracoeliac aorta (with concomitant revascularisation of the coeliac trunk if it is occluded), aortomesenteric endarterectomy, or side-to-side anastomosis between the SMA and aorta. Prosthetic material should be avoided since transmural migration of bacteria is likely to contaminate the graft. Also, if the lower aorta and iliac systems are heavily calcified, then it may be better

to select the supracoeliac aorta for the inflow site of the bypass. The use of a temporary shunt for immediate reperfusion while the bypass is being constructed seems sensible [21].

At this point in the procedure, the viscera need to be inspected again and any dubiously viable bowel resected. If the patient is otherwise young and fit, then it may be better to resect all the necrotic bowel, exteriorise the remaining ends, and consider long-term total parenteral nutrition or small-bowel transplantation. If there is no evidence of arterial compromise and a pulse is palpable in the SMA, then one should suspect either NOMI or MVT.

NOMI usually results in widespread patchy ischaemia. Treatment entails resection of obviously non-viable bowel, attempting to be as conservative as possible. The remaining bowel must be inspected at a second-look laparotomy 24–48 h later. During surgery, several strategies have been described to improve mesenteric flow: a combination of systemic dopamine and an opiate epidural [25], papaverine infusion (30–60 mg/h) into the SMA either via direct puncture fine bore or angiographically placed catheter [13, 26, 27]. At relook laparotomy, the extent of the ischaemia should be reassessed; treatment may need to be abandoned if the gangrene is progressing. Mortality of this condition is depressingly high at 70–80% despite the therapeutic measures outlined above [13].

The classic laparotomy findings in MVT are ascites and swollen omentum with bowel infarction. Like NOMI, MVT is managed by resection of non-viable bowel, which is most frequently sharply demarcated and found in the mid-small bowel. Again, second-look laparotomy is mandatory. Anticoagulation with heparin and then warfarin is mandatory in view of the high incidence of recurrent MVT. Investigation of any underlying prothrombotic disorder along with long-term anticoagulation improves survival [28, 29].

Thrombolysis has been used successfully in MVT in two studies where the diagnosis had been established by non-invasive means and peritoneal signs had not developed [30, 31]. Venous thrombectomy has also been reported to be successful in a handful of cases [19, 32–34]. This procedure is likely to be difficult, as the peritoneum is usually oedematous. **[Q4: B, D]**

The classic features of ischaemia are oedema, loss of peristalsis, loss of surface sheen, staining of the serosa, absent mesenteric pulsation, or frank gangrene with or without perforation (Fig. 25.3). The decision at surgery is, after revascularisation (providing it was appropriate or feasible), how much small bowel to resect. Once there is good flow to the viscera, then reversibly ischaemic segments should declare themselves viable and the rest will need to be resected. Other adjuncts to inspection and palpation are continuous-wave Doppler, pulse oximetry and fluorescein dye [35]. The next question is whether to exteriorise or anastomose the open ends of the bowel. The arguments against exteriorising are that many stomata may be necessary and they do not guarantee that the intervening segments may not subsequently become ischaemic; however, this is extremely safe and avoids the complication of a necrotic anastomosis. Performing primary anastomoses and leaving the abdomen open and covering it with a see-through bag (a cut-open bag of saline) [36] allows direct visualisation of the viscera at all times; second-look laparotomy can then be planned on the appearance of the gut [36]. If all looks well by 72 h and the patient's condition is stabilising, then they can be returned to theatre for planned abdominal closure. **[Q5: A, C, E], [Q6: B, E]**

The short-term management of these cases is demanding on staff and time, but if best results are to be achieved, then no short cuts can be taken.



Fig. 25.3. Gut showing features of fixed staining.

AMI is a treatable vascular emergency. It requires a high index of clinical suspicion, rapid aggressive resuscitation and diagnostic manoeuvres to determine the specific underlying cause. This will allow a prompt, directed revascularisation procedure after optimisation of cardiac performance, or correction of a hypercoagulable state. This effort is directed at maximising the amount of salvageable bowel. These strategies are the cornerstones for a successful outcome in this life-threatening vascular catastrophe.

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26. Renovascular Hypertension

David Bergqvist and Martin Björck

A 58-year-old male smoker had had essential hypertension for about 15 years. It was well balanced with diuretics. He came for regular checks over the years. At such a check 3 months ago, his blood pressure was 195/110 mm Hg. With the addition of a beta-blocker and an angiotensin-converting enzyme (ACE) inhibitor, it was possible to keep the pressure at around 180/100 mm Hg; thus it was not an optimal treatment result. Creatinine had increased over the last 3 months from 90 to 180–200 µmol/l. The patient lived an active life and felt well.

Question 1

Which of the following statements support your suspicion that the patient has renovascular hypertension?

A. The patient has had essential hypertension for more than 10 years.

B. There is difficulty in controlling the blood pressure with three different drugs.

C. There was an increase in serum creatinine when starting ACE inhibitor treatment.

D. There is an absence of an epigastric bruit.

Question 2

What is the best investigation to carry out to decide whether the hypertension has a renovascular origin?

A. Renin in serum.

B. Renography with captopril provocation.

C. Magnetic resonance angiography (MRA).

D. Duplex scanning.

E. Angiography with pressure gradient.

In this patient, an isotope renogram showed a prolonged uptake on the left side after captopril provocation. Duplex investigation showed a peak systolic velocity in the left renal artery of 2.7 m/s with normal findings on the right side. Both kidneys measured about 9 cm in length, the left being perhaps 0.5 cm shorter. MR angiography showed a left-sided ostial stenosis with a suspect post-stenotic dilatation. It was decided to perform an angiography with the aim to establish whether there was a left-sided stenosis, and if there was to give a basis for deciding the optimal treatment. The suspicion of a left-sided stenosis was verified. It was localised near the aortic wall. The narrowest part of the renal artery was about 1.5 mm, with a distinct post-stenotic dilation. A pressure gradient of 40 mm Hg was measured. Corkscrew collaterals were seen along the ureter.

Question 3

What is the first treatment option to normalise the renal artery stenosis?

A. Aortorenal bypass.

B. Thromboendarterectomy.

C. Percutaneous transluminal angioplasty (PTA) with stent.

D. PTA.

E. Nephrectomy.

Question 4

What is the main complication that may occur directly after renal artery PTA?

A. Arterial rupture.

B. Occlusion.

C. Microembolisation.

The patient underwent dilation with a stent without complications and was sent home the day after treatment in good condition. During the next 3 weeks, he was able to stop taking the beta-blocker and ACE inhibitor, and his blood pressure was maintained at around 160/90 mm Hg. Creatinine was around 100 μmol/l.

Question 5

How should the patient be followed up?

A. Serum creatinine.

B. Clinical investigation with blood pressure control.

C. Angiography.

D. Duplex ultrasonography.

E. Captopril scintigraphy.

Commentary

Renovascular disease is responsible for hypertension in around 1 per cent of all patients with high blood pressure. The definition of renovascular hypertension is, however, complicated by the complex and sometimes unclear relation between morphological alterations in the renal artery and the physiological effect of the stenosis. Hypertension is seen in 10–15 per cent of the adult population, and a renovascular cause varies between 0.2 and 5 per cent of those with hypertension; most often, a figure of around 1 per cent is given [1]. The most common cause is arteriosclerosis, which is the probable aetiology in our case (age, sex, smoking, previous hypertension). With the potent antihypertensive drugs of today, it is possible to obtain fairly good blood pressure control in patients with renovascular hypertension. Patients therefore are often not evaluated until there is ischaemic nephropathy with increasing creatinine [2]. As every patient with hypertension cannot be screened for renal artery stenosis, there are some criteria that may raise suspicion:

- Rapid onset of hypertension in young people.
- Rapid deterioration of previously well-controlled essential hypertension.
- Malignant hypertension or hypertensive crises.
- Three-drug-resistant hypertension.
- Hypertension and deteriorated renal function.
- Impaired renal function when starting ACE inhibitors.
- Abdominal, flank or back bruit.

Our patient had had essential hypertension for more than 10 years, there was difficulty in controlling the blood pressure with three drugs, and there was an increase in serum creatinine when starting ACE inhibitor treatment. The absence of bruit is of no value; its presence would have given further support. **[Q1: A, B, C]**

Diagnosis is difficult and the low prevalence contributes to the problem. If there is a clinical suspicion of renovascular hypertension, as in our patient, then renin in serum is of little if any value [3]. Angiography is not indicated to decide whether there is renovascular hypertension: it gives a morphological picture but there are many patients even with advanced renal artery stenosis in whom it is not of functional importance [4–6]. Although hypertension is prevalent in patients with renal artery stenosis, this does not mean it is a causal relationship [7]. To further strengthen the diagnosis a trans-stenotic pressure gradient of >15 mm Hg indicates that stenosis is of functional importance. The only absolutely certain way to define the relationship of renal artery stenosis in hypertension or ischaemic nephropathy

is to observe the beneficial effect of an intervention. MRA and CT angiography are increasingly used to verify the diagnosis. The alternatives, isotope renography, preferably with captopril provocation [8], and duplex ultrasonography [9], both give some functional information but none has sufficiently high sensitivity and specificity to be truly diagnostic. **[Q2: C, E]**

The first treatment choice in an uncomplicated case of renal artery stenosis is PTA [10], which was performed in this patient. Because of an irregular lumen with dissection, the procedure was completed with a stent with good morphological result. Stent placement in renal arteries was first used to correct suboptimal balloon dilations (recoil or plaque resistance), and complications such as dissection or restenosis [11–13]. Some authors recommend primary stenting [11, 14]. **[Q3: D]**

Acute serious complications are infrequent. In a recent overview, the total rate was 17 per cent, with 2 per cent leading to surgery [15]. The most common serious acute complication is acute occlusion. **[Q4: B]**

PTA of the renal artery stenosis is a convenient procedure for the patient, with few complications and short hospitalisation time. However, restenosis is not uncommon [10, 16, 17], and it therefore seems reasonable to have a surveillance programme, although the value of such a programme has not been established in any scientific study. The risk for restenosis is highest during the first year [10]. When there has been adequate preinterventional duplex investigation, as in this patient, it seems logical to continue with this investigation every 3 months during the first year and biannually thereafter. If this is not possible, then clinical investigation with blood pressure control will be the choice. It must be noted, however, that the evidence basis for follow-up routines after renal artery angioplasty has yet to be defined. **[Q5: B, D]**

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27. Management of Portal Hypertension

Yolanda Y. L. Yang and J. Michael Henderson

A 37-year-old woman with a history of hepatitis C, cirrhosis, and esophageal varices presented with hematemesis and melena. The patient had a history of a prior esophageal variceal bleeding episode 7 years ago, which required transfusion of 4 units of packed red blood cells (PRBC) and had been treated with endoscopic sclerotherapy. She was placed on nadolol at that time.

Question 1

If the patient had been found to have varices before any bleeding episode, she would benefit from which of the following?

A. Endoscopic treatment: sclerotherapy or band ligation.

B. Transjugular intrahepatic portal systemic shunt (TIPS).

C. Non-cardioselective beta-blocker.

D. A surgical shunt.

The patient re-presents one year prior to her current admission with a further variceal bleed documented at endoscopy, which required 5 units of PRBC. The acute episode of bleeding was managed with variceal banding, and the patient underwent a course of banding on an outpatient basis. She had no encephalopathy at that time, but did develop some ascites for a short period that responded to salt restriction, Aldactone, and Lasix. Over this past year, her liver function tests have been stable with her bilirubin at 1.0, albumin at 3.5, and a normal prothrombin time.

Question 2

An episode of acute variceal bleeding usually requires which of the following?

A. ICU admission with hemodynamic monitoring, blood, blood products, and fluid resuscitation.

B. An emergency portacaval shunt.

C. A transjugular intrahepatic portal systemic shunt.

D. Endoscopic therapy with sclerosis and/or band ligation.

E. Pharmacologic therapy.

At the present admission the patient is alert and oriented with no evidence of encephalopathy. She has well-preserved muscle mass on examination and is not clinically jaundiced. Her abdomen shows minimal ascites, with no hepatomegaly, but evidence of splenomegaly. Her laboratory studies showed a hemoglobin of 7 g/dl, AST 24, alkaline phosphatase 84, albumin 2.6, bilirubin 3.4, and international normalized ratio (INR) 1.6. She was receiving blood transfusion when examined and octreotide infusion at 50 μg/h. Esophagogastroduodenoscopy showed clot over an esophageal varix with evidence of other non-bleeding varices in both the distal esophagus and gastric fundus.

Question 3

Which of the following studies are important in evaluation and management decisions?

A. Calculation of Child's score.

B. Calculation of MELD score.

C. Endoscopy.

D. Doppler ultrasound.

E. Angiography.

Question 4

Which of the following statements are accurate in prevention of recurrent variceal bleeding?

A. All patients require portal decompression.

B. First-line treatment is with endoscopic band ligation and a beta-blocker.

C. Variceal decompression can only be achieved with a surgical shunt.

D. Liver transplant is good treatment for variceal bleeding in patients with end-stage liver disease.

Question 5

Decompression of gastroesophageal varices:



Fig. 27.1. Splenic artery injection. The catheter is in the splenic artery and is injected with contrast.

A. Can be achieved equally well with surgical shunt or TIPS.

B. Should only be used for patients who have failed endoscopic and pharmacologic therapy for bleeding varices.

C. Improves survival in patients with bleeding varices when compared to endoscopic therapy.

D. Is best achieved by liver transplant for all patients with variceal bleeding.

The patient presented in this case had recurring bleeding episodes through first-line treatment and was therefore a candidate for decompression. Evaluation with angiography and ultrasound showed patent splenic and portal veins and a normal left renal vein (Figs 27.1–27.4). The patient had an elective distal splenorenal shunt for variceal decompression. She was in hospital for 7 days, and was discharged following shunt catheterization (Fig. 27.5) and documentation of patency. Follow-up over the next 4 years showed some progression of her hepatitis C, but no further episodes of variceal bleeding.

Commentary

The case presented illustrates several important points:

- Prophylactic management of gastroesophageal varices, strictly speaking, is prior to the first bleeding episode. The risk of bleeding in a patient with cirrhosis is



Fig. 27.2. Splenic vein. The contrast is followed as it flows out of the splenic vein and then cephalad in the portal vein. There is a significant umbilical vein (double shadow with the portal vein) and a small left gastric vein (off the splenic vein) filling on this study. The second, more caudal catheter is positioned within the left renal vein to aid preoperative determination of the spatial relationship between the splenic and left renal veins.



Fig. 27.3. Normal left renal vein. This study has been performed via the right jugular vein, and demonstrates the left renal vein as it heads cephalad towards the inferior vena cava.



Fig. 27.4. Circumaortic left renal vein. A circumaortic left renal vein, present in 20 percent of the population, does not prevent construction of a distal splenorenal shunt. The superior and anterior component is always larger and can be used for the shunt. More problematic is a totally retroaortic vein, found in 4 percent of the population, which runs transversely and is more fixed in the retroperitoneum, making exposure of the anastomosis more difficult. These patients are better served with a splenocaval shunt.



Fig. 27.5. Postoperative catheterization of the distal splenorenal shunt. The tip of the catheter lies within the mobilized splenic vein, and the first bend marks the splenorenal anastomosis. The skin staples mark the extended left subcostal incision.

approximately 30 percent. Once they have had one bleeding episode, the risk of rebleeding rises to 75 percent without active therapy. Non-cardioseclective beta-blockade with propranolol or nadolol is the preferred treatment for true prophylaxis for medium or large size varices.

- Acute variceal bleeding is an emergency situation with a high mortality if not appropriately managed. Appropriate monitoring, pharmacologic therapy, and endoscopic diagnosis and treatment are the mainstays of treatment of an acute bleeding episode. It is a very small percentage of patients who do not have their bleeding controlled with the above measures and come to an emergency decompression.
- The evaluation of the patient after an acute bleeding episode should assess the varices (endoscopy), the vascular anatomy (ultrasound and angiography) (Figs 27.1–27.4), and the liver disease (Child's class and MELD score). **[Q3: A, B, C, D, E]**
- When a patient has had an acute bleeding episode, their risk of rebleeding is over 70 percent if they have no specific treatment. The initial approach to treatment is to reduce the portal hypertension with a non-cardioselective beta-blocker, and to deal with the bleeding varices directly with endoscopic therapy. The majority of patients do not need variceal decompression at this stage. If the patient obviously has advanced to end-stage liver disease, a transplant evaluation is in order, and appropriate candidates should move forward with that treatment **[Q4: B, D]**
- When patients have recurrent bleeding through first-line treatment they may need decompression of their gastroesophageal varices. Surgical therapy will do this well in 95 percent of patients, while the success rates of radiologic shunts in the literature are not this high. Decompression of varices does not improve the survival of patients compared to other first-line treatment options. Liver transplant provides excellent variceal decompression, but its use is dictated by end-stage disease rather than variceal bleeding. **[Q2: A ,D, E]**

General Considerations

The major complications of portal hypertension are variceal bleeding, ascites, and progressive hepatic dysfunction. Ascites and encephalopathy are signs of decompensation, and as a general guideline, are only effectively managed by liver transplant. Not all patients with these clinical endpoints may be suitable candidates for transplant. In contrast, variceal bleeding can occur in patients who have well-preserved liver function and therefore have a wider range of treatment options available.

The etiology of portal hypertension may be presinusoidal, as in portal vein thrombosis; sinusoidal, as in cirrhosis; and rarely, post sinusoidal, as in Budd–Chiari syndrome. Much the most common etiology in the USA and Europe is cirrhosis, with approximately 90 percent of patients having this etiology. The evaluation of the patient with suspected portal hypertension includes an endoscopy to assess size and extent of varices with risk factors for bleeding. Larger varices with red color signs are at increased risk of bleeding or of rebleeding. Laboratory tests should assess liver function, and overall disease status. Non-specific tests include bilirubin, prothrombin time, albumin, and liver enzymes. Recently documented is the importance of serum creatinine in assessing overall severity of disease and prog-

Table 27.1. Child–Pugh classification

Parameter	1 point	2 points	3 points
Serum bilirubin (mg/dl)	<2	2–3	>3
Albumin (g/dl)	>3.5	2.8–3.5	<2.8
Prothrombin time (↑s)	1–3	4–6	>6
(INR)	<1.7	1.71–2.24	>2.25
Ascites	None	controlled medically	controlled poorly or uncontrolled
Encephalopathy	None	1–2	3–4

Classification: A, 5–6 points; B, 7–9 points; C, 10–15 points.

Table 27.2. MELD score for stratification of liver disease severity

$$\text{Score} = 0.957 \times \log_e \text{creatinine (mg/dl)} + 0.378 \times \log_e \text{bilirubin (mg/dl)} + 1.120 \times \log_e \text{INR}$$

nosis. The two standard methods for assessing this are the Child–Pugh score (Table 27.1), and the Model for Endstage Liver Disease (MELD score – Table 27.2). Other laboratory studies that are important relate to the etiology with hepatitis panels, alpha-fetoprotein as a marker for hepatocellular carcinoma, and specific markers for metabolic diseases such as hemochromatosis and Wilson's disease.

Imaging studies are important in evaluation, with ultrasound used to assess the liver morphology, and Doppler evaluation for liver vasculature. Patency of the main vessels and direction of flow can be assessed well with Doppler ultrasound. Angiography is still indicated for patients being considered for surgery. Accurate assessment of the splenic, portal, and left renal veins is important for distal splenorenal shunt, and may further elucidate details that are not seen on ultrasound. Liver biopsy is occasionally indicated in some patients for clarification of etiology and to delineate the activity of the liver disease process.

Management of portal hypertension falls in to three broad groups:

- Prophylactic treatment.
- Management of an acute variceal bleed.
- Prevention of recurrent variceal bleeding.

Prophylactic treatment is indicated for moderate or large size varices to reduce the risk of an initial bleed. Varices are present in 30–60 percent of patients with cirrhosis. Thirty percent of patients with varices will bleed from them. After an initial bleed, 20–50 percent will rebleed in the first week, and 75–80 percent will rebleed within a year. The mortality of an acute bleeding episode is approximately 25 percent. To reduce the risk of this initial bleed, the goal is to reduce portal pressure to <12 mm Hg or by 20 percent from the baseline. This is best achieved with a non-cardioselective beta-blocker (propranolol, nadolol) [1]. Other treatments, such as endoscopic therapy, TIPS, or surgical shunt are not indicated for prophylaxis. There are currently further ongoing trials looking at band ligation for

patients with large varices where this might be an appropriate method for prophylaxis [2]. **[Q1: C]**

Management of an acute variceal bleed involves resuscitation, pharmacologic reduction of variceal pressure, and endoscopic therapy [3]. Resuscitation requires careful monitoring and enough blood volume and transfusion to maintain blood pressure, but not over-transfuse and precipitate a vicious cycle of further bleeding. Octreotide is the drug of choice for pharmacologic pressure reduction and is given as a continuous infusion of 50 μg/h. Endoscopic therapy is combined with endoscopic evaluation and it best done with banding of varices if visibility is adequate. Occasionally, direct sclerotherapy injection may be required to stop acute bleeding. In the <10 percent of patients who do not have their acute bleeding controlled with such measures, or in whom early significant rebleeding occurs, early decompression is occasionally required. This can best be achieved with TIPS at the current time.

Prevention of recurrent variceal bleeding has to take in to account the risk of rebleeding, and the underlying liver disease. First-line treatment to prevent rebleeding is with a course of endoscopic banding in conjunction with pharmacologic therapy to reduce portal pressure with non-cardioselective beta-blocker [3]. This combination will reduce the risk of rebleeding to approximately 20 percent. Banding has been shown to be considerably better than sclerotherapy in terms of bleeding control and fewer complications. However, mortality is not significantly different in the randomized trials that compared banding to sclerotherapy. Concurrent with this first-line treatment, assessment and management of the underlying liver disease is important. At this time, assessment as to whether the patient is headed for transplant now or in the foreseeable future is important. If this is the case, more invasive therapies are precluded, transplant evaluation should be completed, and the patient should be appropriately listed. In this population, transplant has significantly improved the outcome of patients with Child's class C cirrhosis who have end-stage disease, and have variceal bleeding.

For better-risk patients who have recurrent bleeding through first-line treatment, variceal decompression may be indicated. The current options are with a radiologic shunt (TIPS) [4] or with a surgical shunt such as a distal splenorenal shunt (DSRS) [5], or some type of portacaval shunt. The literature data indicates that the rebleeding rate with TIPS is in the 15–20 percent range. Rebleeding with surgical shunts is in the 5 percent range. However, TIPS can be achieved in a much less invasive fashion compared to the major surgery required for a surgical shunt. Two randomized trials have compared TIPS to surgical shunt. Rosemurgy et al. [6] compared TIPS to an 8-mm H-graft interposition portacaval shunt. They showed notably lower rebleeding in the surgical shunt group, significantly less need for transplant, but no difference in mortality. They concluded that surgical shunt was preferable to TIPS. Henderson et al. [7] have compared TIPS to DSRS in Child's class A and B patients. They showed no significant difference in rebleeding between DSRS (6 percent) and TIPS (9 percent) in this trial; however, the TIPS group had an 82 percent reintervention rate to maintain decompression and this excellent control of bleeding. The encephalopathy rates were not significantly different in the two groups, and neither was survival. The conclusion from this trial is that bleeding can be equally efficaciously managed with TIPS or DSRS with no difference in survival or encephalopathy; however, significantly more reintervention is required in patients managed with TIPS. This trial is summarized in Table 27.3.

Have covered stents improved TIPS outcome? A multicenter prospective randomized trial in Europe [8] has shown a significantly lower dysfunction with

Table 27.3. Data for DSRS versus TIPS randomized trial [7]

Results	DSRS $n = 73$	TIPS $n = 67$	p
Rebleeding	4 (5.5%)	6 (9%)	NS
Reintervention	8 (11%)	55 (82%)	<0.0001
Encephalopathy			
Single event	36 (50%)	34 (50%)	NS
Multiple events	18 (25%)	17 (25%)	NS
Survival			
2-year	81%	88%	NS
5-year	64%	60%	NS

Table 27.4. Data for covered versus uncovered TIPS – European trial [8]

Results	PTFE	Uncovered	p
"Dysfunction"	5 (15%)	18 (44%)	<0.001
Bleeding	2/19 (11%)	4/29 (14%)	NS
Ascites	1/20 (5%)	8/12 (67%)	<0.05
Reintervention	6/39 (15%)	22/41 (54%)	<0.05
Survival	27/39 (69%)	22/41 (54%)	NS

covered stents, with a particular advantage in control of ascites in that trial. Survival was not significantly different with covered or uncovered TIPS stents (Table 27.4).

The data at this time would therefore indicate the following:

- Patients with cirrhosis and moderate to large varices should receive prophylactic therapy with non-cardioselective beta-blocker prior to the initial bleed.
- Patients with acute variceal bleeding should be managed in an intensive care unit with careful monitoring, adequate transfusion, pharmacologic and endoscopic therapy.
- Patients with recurrent variceal bleeding should be managed with endoscopic banding and a non-cardioselective beta-blocker. Only those patients who have well-preserved liver function, and rebleed through first-line treatment, should be considered for decompression. This can be achieved with either a surgical shunt or TIPS. Patients with end-stage liver disease need to be evaluated for their suitability for transplant and transplanted if appropriate. **[Q5: B]**

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28. Management of Patients with Carotid Bifurcation Disease

Wesley S. Moore

A 72-year-old white male was referred for evaluation and management following the finding of an asymptomatic carotid bruit, picked up on routine physical examination by his primary-care physician. The patient was asymptomatic with respect to ocular or hemispheric ischaemic events. His risk factors included a 30-year history of smoking one pack of cigarettes a day, which he quit a year ago. He had hypertension that was controlled well by two drugs. He had no history of coronary artery disease, diabetes mellitus, or symptoms of peripheral vascular disease. On physical examination, his temporal pulses were equal. His carotid pulses were full and equal, but there was a loud bruit over the right carotid bifurcation. His femoral, popliteal, dorsalis paedis and posterior tibial pulses were normally palpable bilaterally.

Question 1

What should the next step in this patient's evaluation be?

A. Counselling with respect to the nature of carotid ischaemic attacks.

B. Start the patient on an antiplatelet drug such as aspirin.

C. Counsel the patient with respect to the importance of refraining from cigarette smoking and careful control of blood pressure.

D. Obtain bilateral carotid duplex scanning.

E. All of the above.

The patient underwent a bilateral carotid duplex scan. **[Q1: D]** The scan demonstrated a category 60–79% right carotid bulb stenosis. The plaque characteristic was one of mixed consistency, a mildly irregular surface, and minimal calcification. The left carotid bulb showed a category 20–59% stenosis. Both vertebral arteries were imaged with normal antegrade flow velocities.

Question 2

What would be appropriate management for this patient?

A. Elective carotid endarterectomy.

B. Full Coumadin anticoagulation.

C. Aspirin antiplatelet management and risk factor control.

The patient was placed on aspirin antiplatelet therapy, counselled regarding the importance of good blood pressure control, and given an appointment for a return visit in 6 months' time for a repeat carotid duplex scan to see whether there was any evidence of progression. The patient was also counselled regarding the importance of calling the vascular service should he develop ocular or hemispheric transient ischaemic attacks within the 6-month interval before his return appointment. **[Q2: C]**

The patient did quite well for the next 4 months; then one afternoon, he noted the onset of an episode of numbness and weakness of his left hand. The hand was not totally paralysed, but it was clearly numb, weak and uncoordinated. This cleared completely within a period of 10 min. The patient thought that this might have been related to his arm position and chose to do nothing further until the next day, when the same event occurred. At this point, he called his physician and was advised to return immediately. An emergent carotid duplex scan was ordered. The scan now showed progression to a category 80–99% stenosis with plaque once again of mixed consistency.

Question 3

What is the best management for this patient?

A. Clopidogrel antiplatelet therapy.

B. Full Coumadin anticoagulation.

C. Schedule elective carotid endarterectomy 1 month from now.

D. Urgent right carotid endarterectomy.

The patient now had two clear indications for proceeding with carotid endarterectomy: the onset of symptoms in the territory of the carotid lesion, and progression of the lesion to an 80–99% stenosis. Two additional decisions also had to be considered: the timing of operation and whether brain imaging was indicated. In view of the fact that the patient had an appropriate carotid artery lesion, and the symptoms were typical for hemispheric transient ischaemic events in the distribution of the carotid lesion, information gained from brain imaging such as computed tomography (CT) or magnetic resonance imaging (MRI) would be limited. Therefore, the cost/benefit ratio for brain imaging was clearly unfavourable. The timing of carotid endarterectomy was urgent. The patient had a new onset of transient ischaemic attacks and evidence of plaque progression. Therefore, the patient

was now at highest risk of a hemispheric stroke. The optimum management for this patient would be emergent admission to the hospital and rapid evaluation for operation, including the patient's cardiac status. While this was taking place, it would be appropriate to start the patient on intravenous heparin anticoagulation. Once cleared from a cardiac standpoint, plans should be made to proceed with operation either that day or the next morning. **[Q3: D]**

The patient was admitted as an emergency to the hospital and started on intravenous heparin with a loading dose of 5,000 units and a continuing dose of 1,000 units/h. He was seen in cardiology consultation, and an electrocardiogram (ECG) was obtained. In the absence of any symptoms of coronary disease, and with a relatively normal ECG, he was cleared for operation.

Question 4

What should the next step in this patient's management be?

A. Aortic arch angiogram with selected carotid arteriograms.

B. Magnetic resonance angiogram (MRA).

C. CT angiogram.

D. Proceed with operation on the basis of a duplex scan of diagnostic quality in an accredited laboratory.

The patient was taken to the operating room the next morning. Before this, EEG electrodes were placed for intraoperative monitoring. An arterial line was placed for blood pressure monitoring, and general anaesthesia was administered. A vertical incision along the anterior border of the sternomastoid muscle was made. The facial vein was divided, and the common carotid, carotid bifurcation, internal and external carotid arteries were fully mobilised. There was a posterior plaque present in the common carotid artery, which was nonocclusive. The major plaque build-up was in the bulb of the internal carotid artery, which went a short distance beyond the bulb into the internal carotid artery distally. Beyond this point, the vessel was circumferentially soft. The distal internal carotid artery was somewhat collapsed, and no distal pulse was noted. Since the patient had experienced only transient symptoms and not a completed stroke, it was our plan to use an internal shunt only if there were electroencephalogram (EEG) changes with trial clamping. A bolus of 5,000 units of heparin was administered, and the internal, external and common carotid arteries were clamped. The EEG was observed: there were no changes. The amplitude and frequency of the EEG wave form were maintained. A longitudinal arteriotomy was made in the common carotid artery and extended through a very tight carotid stenosis. The plaque within the carotid bulb showed evidence of recent intraplaque haemorrhage. As we passed through the plaque, we emerged into an unencumbered internal carotid artery distally. A bifurcation endarterectomy was then performed with clean endpoints in the internal, external and common carotid arteries. The intimectomised surface was then irrigated with heparinised saline, and small bits of medial debris were removed carefully. Fixation of the endpoint was carried out. Once we were

satisfied that there was no evidence of intimal flap and all of the loose bits of medial debris were removed, attention was turned to closure.

Question 5

Closure of the arteriotomy should be:

A. A primary, carefully placed closure with 6-0 prolene.

B. Closure with a patch angioplasty.

The patient's arteriotomy was closed with a patch angioplasty using a collagen-impregnated knitted Dacron patch that was cut to length and bevelled at each end. Upon completion of the closure, blood flow was begun first to the external then to the internal carotid artery. Excellent pulsation in all vessels was noted. We then carried out a completion angiogram by placing a small needle in the patch and injecting contrast into the carotid bifurcation using a portable cine-fluoro unit. The carotid bifurcation was imaged, and there was an excellent technical result with no evidence of residual stenosis or intimal flap. Intracranial imaging was also carried out, and excellent flow into the carotid siphon and the anterior and middle cerebral arteries was confirmed. After meticulous haemostasis was achieved, a 7.0-mm Jackson Pratt drain was placed in the wound and brought out through a separate stab wound. The platysmal layer was closed with an absorbable suture, and the skin was closed with a subcuticular absorbable suture. An adhesive plastic dressing was applied directly to the skin, and the patient was returned to the recovery room. The patient awoke at his neurological baseline with no evidence of cerebral or cranial nerve deficit. His blood pressure was monitored carefully and was noted to be stable at 150/80 mm Hg.

Question 6

After an appropriate stay in the recovery room, to where should the patient be transferred?

A. An intensive care unit with continual monitoring overnight.

B. A step-down unit with 3 : 1 nursing coverage and monitoring capability.

C. The patient should be left in the recovery room overnight.

D. A regular hospital room.

Since the patient was neurologically intact and was maintaining his normal blood pressure, he was transferred to a regular hospital room for routine overnight care. The patient spent an uneventful night in a regular hospital room. The following morning, we removed the dressing and drain. The patient was ambulatory and on a regular diet and was discharged home on the first postoperative day [1]. This management is typical of the so-called "fast-track" manage-

ment of carotid bifurcation disease. Patients are usually admitted electively on the morning of operation, undergo carotid endarterectomy, spend a period of 2–3 h of observation in the recovery room, transfer to a regular hospital room, and are discharged the following morning. Thus, carotid endarterectomy has become extremely cost-effective in the overall medical economic environment. The patient was instructed to return for a routine visit in 3 weeks. At that time, we obtained a right carotid duplex scan to confirm the result of carotid endarterectomy and to establish a new baseline for future comparison. The next visit will be in 6 months, at which time a bilateral carotid duplex scan will be performed. The objective will be to look for evidence of intimal hyperplasia and recurrent stenosis on the side of operation as well as to document whether there is any progression of disease on the contralateral, nonoperative side. If that test is unremarkable, then the next study will be at the 1-year anniversary. We will then see the patient on a yearly basis and obtain a bilateral carotid duplex scan as a part of that visit.

Commentary

Many decisions concerning recommendation to perform carotid endarterectomy are based upon the degree of stenosis, as measured by a percentage, in the carotid artery. All of the randomised trials have reported their data and have established a baseline threshold stenosis as an appropriate indication for carotid endarterectomy. While this would appear to be a very tangible and straightforward method of quantifying a carotid stenosis, confusion has developed because there are at least two different techniques for measuring percent of carotid stenosis: the North American method and the European method. The North American method was first described in a publication by Blaisdell et al. as part of the Extracranial Arterial Occlusive Disease Study of the 1960s. This method was used in the Veterans Administration Asymptomatic Carotid Stenosis trial and the Asymptomatic Carotid Atherosclerosis Study (ACAS), and was subsequently adopted by North American Carotid Endarterectomy Trial (NASCET) as their method of measurement. The North American method utilises the following formula: percentage stenosis = $1 - R/D$, where R is the minimal residual lumen diameter in millimetres, and D is the diameter of the normal internal carotid artery, distal to the bulb, where the walls of the artery become parallel. In contrast, the European method, which has been used in European trials, including the European Carotid Surgery Trial (ECST) trial, uses the following formula: percentage stenosis = $1 - R/B$, where R again is the minimal residual lumen diameter in millimetres, and B is the projected diameter of the carotid bulb. Since the bulb is not visualised on a carotid arteriogram of a patient with carotid stenosis, a theoretical line is drawn outlining the bulb, emphasising the atheromatous burden within the bulb. Because of these two different methods, percentage stenoses as expressed in the European literature are not equal to percentage stenosis as measured by the North American method. For example, a 60 per cent stenosis European is equal to an 18 per cent stenosis North American; 70 per cent stenosis European equals 40 per cent stenosis North American; 80 per cent stenosis European equals 61 per cent stenosis North American; and 90 per cent stenosis

European equals 80 per cent stenosis North American. Thus, when reading a specific article relating to carotid stenosis, it is important to determine which method of measurement is used in order to appropriately follow the recommendations made by the authors.

The management of patients with asymptomatic high-grade carotid stenosis has been controversial. However, with the recent publication of the ACAS, the approach to management of patients who are asymptomatic has received more universal acceptance in the USA, although not necessarily so in Canada and Europe. The findings of this trial demonstrated that there was a 53 per cent relative risk reduction of stroke in patients who underwent carotid endarterectomy for lesions producing at least a 60 per cent diameter-reducing stenosis, by angiography, when compared with medical management alone. It was also pointed out that a 60 per cent diameter-reducing stenosis by angiography is not the same as a 60 per cent stenosis as measured by duplex scan, since the duplex scan criteria for stenosis are concerned with carotid bulb measurement rather than a stenosis as compared with the diameter of the distal internal carotid artery. It is generally accepted that a 60 per cent diameter-reducing stenosis of the internal carotid artery, by angiography, usually corresponds to a duplex scan finding of an 80–99% stenosis [2].

Patients with carotid artery disease who develop symptoms of hemispheric or monocular transient ischaemic events, or who have had a stroke with good recovery, are clearly good candidates for carotid endarterectomy providing that they have a diameter-reducing stenosis of 50 per cent or greater by angiography. This is now accepted uniformly and has been well established by prospective randomised trials in both North America and the UK [1, 3, 4].

The work-up of patients with carotid bifurcation disease for operation usually involves the performance of a contrast angiogram to confirm the lesion, establish the degree of stenosis, and evaluate the intracranial circulation for other pathology, such as a stenosis of the carotid siphon or an aneurysm of the intracranial branches. As the quality and accuracy of carotid duplex scanning has improved in accredited laboratories throughout the world, the practice of using carotid duplex scan data as the sole imaging requirement before endarterectomy has proliferated. In our own unit, the accuracy of carotid duplex scanning in our laboratory is continually compared with the operative findings at the time of carotid endarterectomy. Initially, the carotid duplex scan data were compared with angiography. As our level of comfort with carotid duplex scanning has increased, contrast angiography has essentially been eliminated in our protocol. The only time we resort to a contrast angiogram is when the carotid duplex scan data and the clinical picture fail to correlate. If the patient has equal upper-extremity blood pressures, as well as good and equal quality pulses in the carotid artery bilaterally, then the likelihood of the patient harbouring a lesion at the level of the aortic arch is quite small. The only other pathology that might be missed in the absence of a contrast carotid angiogram is the rare occurrence of an intracranial lesion. It has been our practice to carry out completion angiography following carotid endarterectomy on the operating table. When the completion study is performed, we always make an effort to examine the intracranial circulation as well. To date, after many hundreds of carotid endarterectomy without angiography, there have only been two instances in which significant intracranial arterial pathology has been found. One was a small intracranial aneurysm measuring less than 10 mm; the other was a siphon stenosis, which, had it been known preoperatively, would not have changed the indication

for carotid endarterectomy. Based upon this experience, we routinely carry out carotid endarterectomy on the basis of duplex scan alone. However, this duplex scan must be performed in our own laboratory, as we are unwilling to accept data from other laboratories as the sole basis for proceeding with operation. While there are many excellent laboratories that provide reliable data, we routinely cross-check data from outside laboratories with a test in our own laboratory. Since duplex scanning is relatively inexpensive, and since it has become a substitute for expensive studies associated with morbidity and mortality, such as contrast angiography, it is our opinion that this additional cost is money well spent [5]. Contrast angiography, while a longstanding gold standard, is expensive, promotes patient anxiety, and is associated with neurological morbidity and mortality. In the ACAS, where angiography was required before carotid endarterectomy, the risk of the angiogram with respect to stroke morbidity and mortality was equal to the risk of the operation itself [1]. MRA, while noninvasive, tends to be less accurate than a well-performed carotid duplex scan. MRA of the carotid bifurcation will frequently overestimate the percentage of stenosis and will lead to unnecessary operation in many instances. CT angiography, while more accurate, requires a large intravenous contrast bolus to perform the study. **[Q4: D]**

Another controversy in the management of patients with carotid bifurcation disease concerns the question of whether a carotid arteriotomy should be closed primarily or with a patch angioplasty. For many years, we routinely closed arteriotomies primarily when the vessel appeared to be of good calibre. A retrospective review of our data suggested that this had been a good practice in that our incidence of restenosis had been quite low. Many retrospective comparisons as well as prospective trials have shown inconclusive data concerning the merit of patch angioplasty versus primary closure. However, recently, a prospective trial in patients scheduled for staged bilateral carotid endarterectomy in whom one side was primarily closed and the second side closed with patch angioplasty conclusively demonstrated that those sides closed with patch angioplasty were associated with a statistically lower incidence of restenosis and complication. Based upon these convincing data, it is now our practice to routinely close all arteriotomies with a patch angioplasty [6]. **[Q5: B]** Other surgeons have modified their surgical practice to perform the operation using eversion endarterectomy, thus avoiding a longitudinal arteriotomy. For those surgeons who are experienced with this technique, and in properly selected patients, this also appears to be a satisfactory alternative. The postoperative monitoring of the patients is important in ensuring the best outcome for these patients. In the past, it had been our practice to monitor patients routinely in the intensive care unit. However, with a retrospective review of our experience, the likelihood of having an untoward event requiring intensive-care nursing in a patient who was neurologically intact and with a normal blood pressure was extremely low. Therefore the cost/benefit advantage of intensive care unit utilisation was clearly not there. We now routinely send patients to a regular hospital room. To date, there have been no untoward incidents that have led us to regret this policy. **[Q6:D]**

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29. Carotid Endarterectomy and Cranial Nerve Injuries

Christos D. Liapis and John D. Kakisis

A 75-year-old male was admitted with a high-grade left internal carotid artery (ICA) stenosis. The patient had a history of a reversible ischemic neurological defect (RIND) causing him a right upper- and lower-extremity paralysis and dysarthria a month prior to his admission. Colour duplex ultrasonography revealed a carotid bifurcation stenosis with a 90 per cent reduction in the diameter of the left ICA. The right ICA was also stenosed with an 80 per cent diameter reduction. There was no history of cerebrovascular symptoms that could be attributed to the right ICA stenosis.

On examination there were bilateral carotid bruits. Peripheral pulses were normal in the upper extremities. In the lower extremities there were no pulses distal to the popliteals. Full blood count, biochemical profile and a clotting screen were within normal limits, while an electrocardiogram (ECG) and dipyridamole stress test revealed changes consistent with coronary artery disease. Chest X-ray and arterial blood gas were normal. Computed tomography (CT) scan of the brain showed a small cerebral infarct at the left parietal lobe and two smaller ones at the right.

Question 1

What other preoperative examination would you perform routinely before taking this patient to the operating theatre for a carotid endarterectomy?

A. Lumbar puncture.

B. Electroencephalography (EEG).

C. Evaluation of the function of the IXth, Xth and XIIth cranial nerves.

D. Ocular plethysmography.

E. Magnetic resonance imaging (MRI) of the brain.

A preoperative examination of the IXth, Xth and XIIth cranial nerve function was carried out, which revealed no neurological abnormalities.

Before the operation, the surgeon had a detailed discussion with the patient, including information about the possible complications of carotid endarterectomy.

Question 2

Which of the following is not an expected complication following carotid endarterectomy?

A. Stroke.

B. Myocardial infarction (MI).

C. Trigeminal nerve palsy.

D. Vocal cord paralysis.

E. Hypoglossal palsy.

Following informed consent, the patient underwent a left carotid endarterectomy. Postoperatively, neurological examination was normal, but the patient complained of hoarseness.

Question 3

Which of the following is the most likely cause of speech malfunction in our patient?

A. Injury to the vocal cords during intubation.

B. Recurrent laryngeal nerve injury.

C. Superior laryngeal nerve injury.

D. Injury to the larynx due to excessive retraction.

E. Cerebral haemorrhage.

Indirect laryngoscopy revealed left vocal cord paralysis. The postoperative course was otherwise uneventful. The patient was discharged on the fourth postoperative day.

Question 4

How long after the operation should the patient be re-evaluated, considering his vocal cord paralysis?

A. Weekly.

B. Monthly.

C. At 2 and 6 months.

D. There is no reason for re-evaluation since all of these palsies resolve within a few days.

E. There is no reason for re-evaluation since no improvement is anticipated.

Two months postoperatively, laryngoscopy and voice evaluation tests revealed normal function of the cranial nerves. The patient was scheduled for right carotid endarterectomy.

Question 5

What are the treatment options for a patient with recurrent laryngeal nerve injury after carotid endarterectomy who needs a contralateral carotid operation?

A. Carotid stenting.

B. Carotid endarterectomy after nerve function recovery.

C. Immediate carotid endarterectomy.

D. Carotid endarterectomy after two weeks.

E. Never operate on this carotid.

Commentary

Carotid endarterectomy is associated with a number of serious complications, including stroke, death, MI, wound complications, hypertension or hypotension, and cranial nerve injuries. **[Q2: C]** Stroke has undoubtedly been the main focus of interest, while local injuries to the cranial nerves and their branches have received considerably less attention. However, cranial nerve injuries during carotid endarterectomy are quite frequent and potentially serious; they may even be life-threatening when they are bilateral.

There is remarkable discrepancy regarding the occurrence of cranial nerve injuries following carotid arterial surgery in various studies. The reported incidence ranges from 3 per cent to more than 50 per cent [1–20] (Table 29.1). These differences are due mainly to the different investigative methods used for the evaluation of the cranial nerve function. The inclusion of speech therapists and their examination tests in the evaluation, as proposed by Liapis et al. [9], can clarify most accurately the function of the nerves in question. An additional problem is the lack of standardisation in reporting lesions, concerning the number of cranial nerves evaluated and the different methods of reporting multiple injuries. Some authors use the number of damaged nerves in their reports, while others use the number of patients with damaged nerves. Nevertheless, the actual incidence of cranial nerve injuries seems to range between 8 and 16 per cent in most reports.

The nerves that can be damaged during carotid endarterectomy, due to their close relation to the carotid bifurcation, are the hypoglossal, recurrent laryngeal,

Table 29.1. Incidence and persistence of cranial nerve injuries after carotid endarterectomy (incidence/persistence). Updated

Ref.	Hypoglossal	Recurrent laryngeal	Superior laryngeal	Marginal mandibular	Glossopharyngeal	Total
1	13.5/0	5.8/0		5.8/0		25/0
2	5.5/0	4/0	1/0	1/0		11.5/0
3	4.7/0	25.6/13.9				30.3/13.9
4	11/4	35/15			1.5/0	39/19
5	3.7*	2.5*		2.2*		8.6*
6	10.7/0.2	1.2/0.2	0.3/0		0.3/0	12.5/0.3
7	5.4/0.8	5.8/0.8	2.1*	2.5*		15.8/2.1
8	2.3/0	3.9/3.1		3.1/0.8		9.3/3.9
9	20/2.5	27.5/5		5/0	0/0	52.5/7.5
10	4.8*	6*		2.4*		13.5*
11	3.3/0	2.6/0		0.4/0	0.7/0	5.6/0
12	8.2/2.5	5.1/0		3.2/1.3		16.4/3.8
13	1.2/0.2	1.2/0.7		0.5/0.2	0.2/0	3/1.2
14	4.4/0.6	7.7/0.6		1.1/0		14.2/1.1
15	8.3/0	3.7/1.8		2.8/0		12.8/1.8
16	2.6/0	1/0		1/1		4.7/1
17	8.6/2.6	3.7/0.6		6.2/2.8	0.4/0	18.9/6
18	4/0.5	7/1		12/1		27/7
19	2.1/0.2	1/0.06		1.8/0.06		5.1/0.5
20	1.1/0	0.6/0.6		3.9/0		5.6/0.6

*Incidence of cranial nerve injury (%).

superior laryngeal and glossopharyngeal nerves, the marginal mandibular branch of the facial nerve, and the greater auricular and cervical sensory branches (Fig. 29.1).

The hypoglossal (XIIth) nerve descends into the neck between the internal carotid artery and the internal jugular vein. It then crosses the internal and the external carotid artery approximately 2–4 cm above the carotid bifurcation to enter the root of the tongue. It is a purely motor nerve, innervating all of the intrinsic muscles of the tongue and three extrinsic muscles: the styloglossus, hyoglossus and genioglossus. The function of the nerve is assessed by measuring tongue muscle activity. Patients are asked to engage in tasks requiring tongue strength, mobility and range of motion, namely to stick out their tongue, to move it from side to side, to raise it to the nose or lower it to the chin and to lick their lips [9]. Injury to the hypoglossal nerve, which can happen during dissection of a high bifurcation, can be avoided by following the ansa hypoglossi up to its junction with the hypoglossal nerve and by dividing the sternocleidomastoid artery and vein separately, since the nerve may be adherent to the posterior surface of these vessels [7]. Division of the ansa hypoglossi, which supplies motor innervation to the infrahyoid muscles, will not be clinically overt. However, it should be spared if possible, because it can be invaluable in cases of future laryngectomy/laryngoplasty. In contrast, injury to the hypoglossal nerve is a serious complication, causing deviation of the protruding tongue to the ipsilateral side, while the relaxing tongue deviates to the contralateral side. Inarticulate speech and clumsy mastication will also ensue, whereas bilateral injury may cause life-threatening airway obstruction.

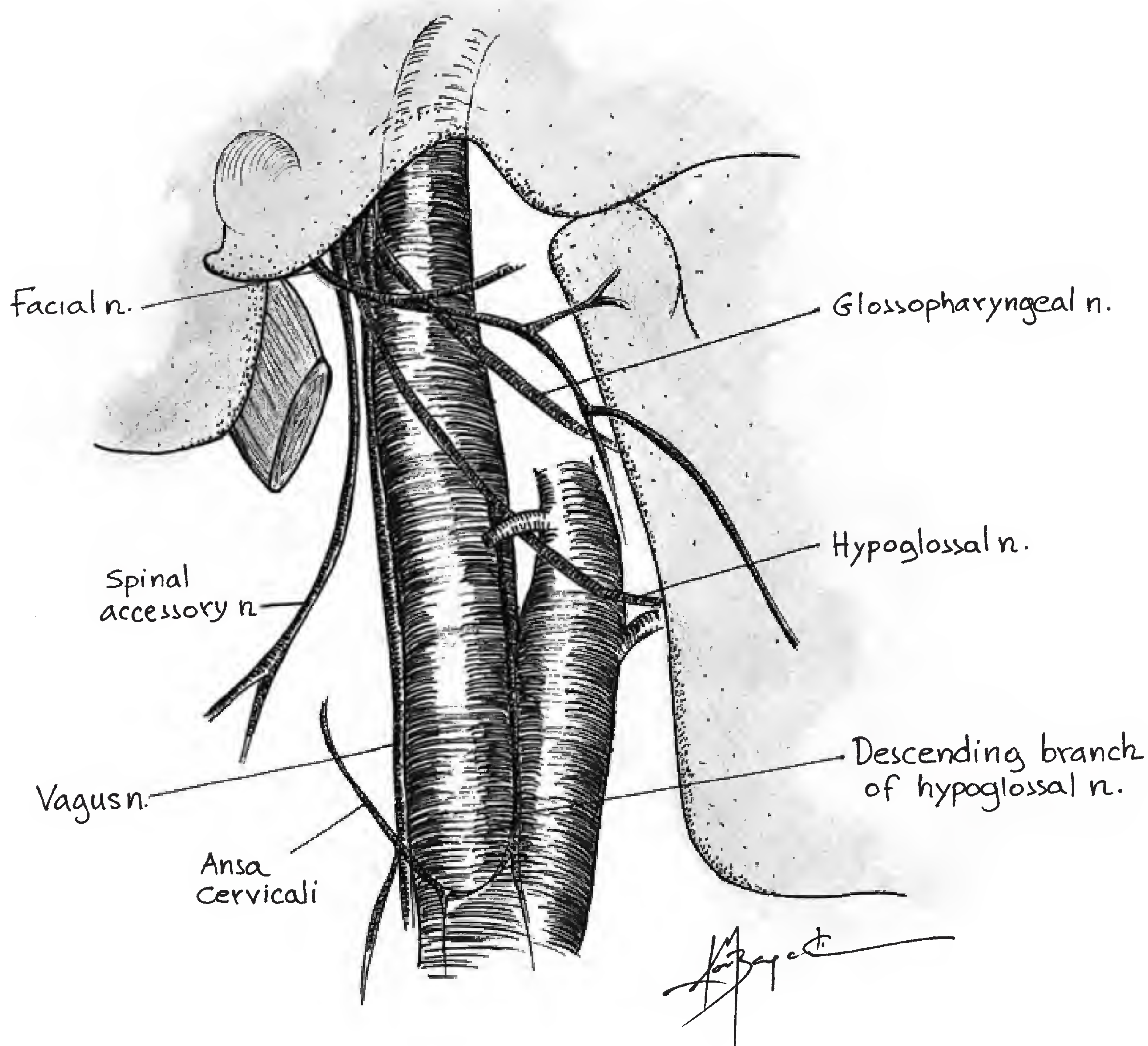


Fig. 29.1. Anatomy of the cranial nerves in relation to the carotid bifurcation.

The superior laryngeal nerve arises from the vagus (Xth) nerve near the jugular foramen and passes diagonally behind the internal and external carotid artery before dividing into an internal sensory branch, supplying sensation to the mucosa of the larynx and an external motor branch, supplying innervation to the cricothyroid muscle and the inferior pharyngeal constrictor. Assessment of the superior laryngeal nerve is done by asking the patient to produce tones of the upper range for about 15 s [9]. Injury to the superior laryngeal nerve can occur if the clamp is placed away from the origin of the external carotid artery and will cause voice fatigue and loss of high-pitch phonation.

The recurrent laryngeal nerve arises from the vagus nerve in the mediastinum, passes around the subclavian artery on the right side and the aortic arch on the left, and ascends the neck in the groove between the oesophagus and the trachea. Only in cases of anomalous origin or course in the neck can the recurrent laryngeal nerve be injured directly during carotid endarterectomy. Dysfunction of the recurrent laryngeal nerve is due to direct injury of the vagus nerve during clamp placement, except in the very rare cases of the non-recurrent laryngeal nerve, when it can be

mistaken for the ansa hypoglossi and severed erroneously. The recurrent laryngeal nerve is motor to all of the muscles of the larynx except for the cricothyroid muscle, and sensory to the mucosa below the vocal cords. Assessment of its function can be made by laryngoscopy as well as by asking the patient to produce sounds of the lower range [9]. In cases of recurrent laryngeal nerve injury, laryngoscopy will reveal paralysis of the ipsilateral vocal cord in a median or paramedian position, resulting in hoarseness and loss of effective cough mechanism. However, it should be noted that postoperative hoarseness does not necessarily imply damage to the recurrent laryngeal nerve. Injury of the vocal cords during intubation, with subsequent oedema or haematoma formation, is a fairly frequent cause of postoperative hoarseness [18, 20]. **[Q3: A]** Differential diagnosis will be made by indirect laryngoscopy.

It should also be noted that vocal cord paralysis of degenerative aetiology is sometimes seen in elderly people. It is therefore recommended that all people scheduled for carotid endarterectomy should undergo preoperative examination of the vocal cords by indirect laryngoscopy. **[Q1: C]** Such an examination is of the utmost importance in patients with a history of thyroidectomy or contralateral endarterectomy. Preoperative evaluation should be completed with examination of the IXth and XIIth cranial nerves in order to document possible pre-existing cranial nerve deficits.

The glossopharyngeal (IXth) nerve exits the skull through the jugular foramen, passes between the internal jugular vein and the internal carotid artery, and then crosses the distal cervical segment of the internal carotid artery and the stylopharyngeal muscle to enter the pharynx. The glossopharyngeal nerve supplies (1) sensation from the posterior third of the tongue and the walls of the pharynx, being the afferent limb of the gag reflex, (2) taste sensation from the posterior third of the tongue, and (3) innervation to the stylopharyngeal muscle. The function of the glossopharyngeal nerve is assessed by administering tests requiring stylopharyngeal muscle activity, such as swallowing, gag reflex and clearing the throat [9]. Damage to the glossopharyngeal nerve can occur when using cautery during high dissection, especially when the posterior belly of the digastric muscle is divided [21]. Difficulty in swallowing is the principal manifestation of such an injury.

The marginal mandibular branch of the facial (VIIth) nerve emerges onto the face from the anterior border of the parotid gland, loops down over the ramus of the mandible, crosses the masseter muscle and enters the perioral muscles in the lower lip. It supplies the risorius muscle and the depressor of the lower lip. Injury to the marginal mandibular branch of the facial nerve may occur during superior extension of the incision and upward retraction. In order to avoid such an injury, the upper end of the incision should be curved posteriorly toward the mastoid process, while the retractors should be placed away from the angle of the mandible and superficial to the platysma [14]. Injury to the marginal mandibular nerve will cause sagging of the ipsilateral corner of the lower lip, which has to be differentiated from stroke.

The spinal accessory (XIth) nerve arises from the jugular foramen, courses posteriorly and passes laterally to the jugular vein, sending branches to the sternocleidomastoid, trapezius and rhomboid muscles. It is not in the field of a routine carotid endarterectomy and can only be injured when dealing with extremely distal internal carotid artery lesions. Paralysis of the spinal accessory nerve results in an inability to shrug the shoulder and rotate the head.

Damage to sensory nerves deriving from the cervical plexus, such as the greater auricular and the transverse cervical nerves, is often thought to be trivial by the sur-

geons performing the endarterectomy, but it may cause considerable annoyance to the patient. Dysfunction of these nerves has been observed in as many as 70–90 per cent of patients in some reports [1, 3]. The greater auricular and the transverse cervical nerves emerge from behind the posterior border of the sternocleidomastoid muscle and course anteriorly over its surface at the upper and lower part of the incision for the carotid endarterectomy, respectively. The greater auricular nerve supplies the skin of the scalp over the mastoid process and the external ear, while the transverse cervical nerve supplies the anterior cervical triangle. Injury to these nerves during the incision or due to excessive traction will result in numbness or painful paraesthesia in the distribution of these nerves.

In the great majority of cases, cranial nerve injuries are caused by blunt trauma, due to excessive retraction. Less common causes are injuries by forcers, electrocautery or the application of arterial clamps. Therefore, most of the cranial nerve injuries are transient and complete recovery occurs within 6 months and usually within 4–6 weeks [9, 11, 14, 18]. Thus, follow-up examination is recommended at 2 months to verify recovery and, in cases of persistent paralysis, at 6 months. **[Q4: C]** Extended follow-up will identify the small subset of patients with delayed (>6 months) complete nerve recovery.

The only factor that has been associated with an increased risk of cranial nerve injury is the duration of operation, with every 30 minutes of operative time increasing the risk of nerve injury by 50%, according to a post hoc analysis of data from the ECST [19]. It seems that the longer a nerve is mechanically retracted, the greater the risk of a traction injury.

Since bilateral injury to the recurrent laryngeal nerves or the hypoglossal nerves is a life-threatening complication, endarterectomy of the contralateral carotid artery should be postponed until cranial nerve dysfunction has resolved (usually after 2 months), unless there are compelling reasons to do otherwise [15].

Endoluminal treatment of carotid stenosis is currently evaluated against carotid endarterectomy. One of the advantages of carotid angioplasty is that it is free of local complications at the neck, including haematomas and cranial nerve injuries. **[Q5: A, B]** According to the CAVATAS trial, cranial nerve palsy occurred in 9 per cent of the surgical patients but not in the endovascular group [22]. Such injuries should therefore be assessed carefully and quoted in all cases of carotid endarterectomy to provide a standard to which emerging catheter-based therapies for carotid stenosis should be compared.

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30. Paragangliomas of the Head and Neck

Johanna G. H. van Nes, Sylvia C. de Jong, Marc R. H. M. van Sambeek and Hero van Urk

A 47-year-old man was referred for investigation and management of a swelling in the neck on the right side (Fig. 30.1). For several months there had been a slowly enlarging and painless palpable mass. Besides problems with swallowing, there were no other symptoms or complaints. The previous medical history was unremarkable and there were no similar lesions known in family members.

On physical examination, the patient had a non-tender mass located just anterior to the sternocleidomastoid muscle in the anterior triangle of the neck. The mass was mobile in a back-forwards direction, but could not be moved in the cranial-caudal direction. No signs of cranial nerve deficits were detected. An echo-duplex showed a highly vascularised structure adherent with the carotid artery located in the bifurcation.

Question 1

Which of the following is the most likely diagnosis causing this patient's swelling?

A. Enlarged lymph node.

B. Goitre of the right thyroid lobe.

C. Paraganglioma.

D. Carotid artery aneurysm.

E. Thyroid tumour.

F. Bronchial cleft cyst.

Based on the history, physical examination and echo-duplex, the diagnosis of a carotid body tumour was made.



Fig. 30.1. Patient with a swelling in the neck, which was diagnosed as a carotid body tumour.

Question 2

Which further examination is preferable to confirm the diagnosis of paraganglioma? Rank them in order and explain why.

A. MRI/MR angiography.

B. Somatostatin receptor scintigraphy (OctreoScan).

C. Angiography.

D. MIBG scanning.

E. Duplex scanning.

F. Needle biopsy.

G. Incision biopsy.

The diagnosis of carotid body tumour was confirmed by angiography (Fig. 30.2) and somatostatin receptor scintigraphy (Fig. 30.3). This showed a tumour of $4 \times 2.5 \times 1.0$ cm. Because of the size and difficulties with swallowing, it was decided to start treating the patient.

Question 3

What are the treatment options?



Fig. 30.2. Angiography of a carotid body tumour showing the typical tulip configuration at the site of the bifurcation.

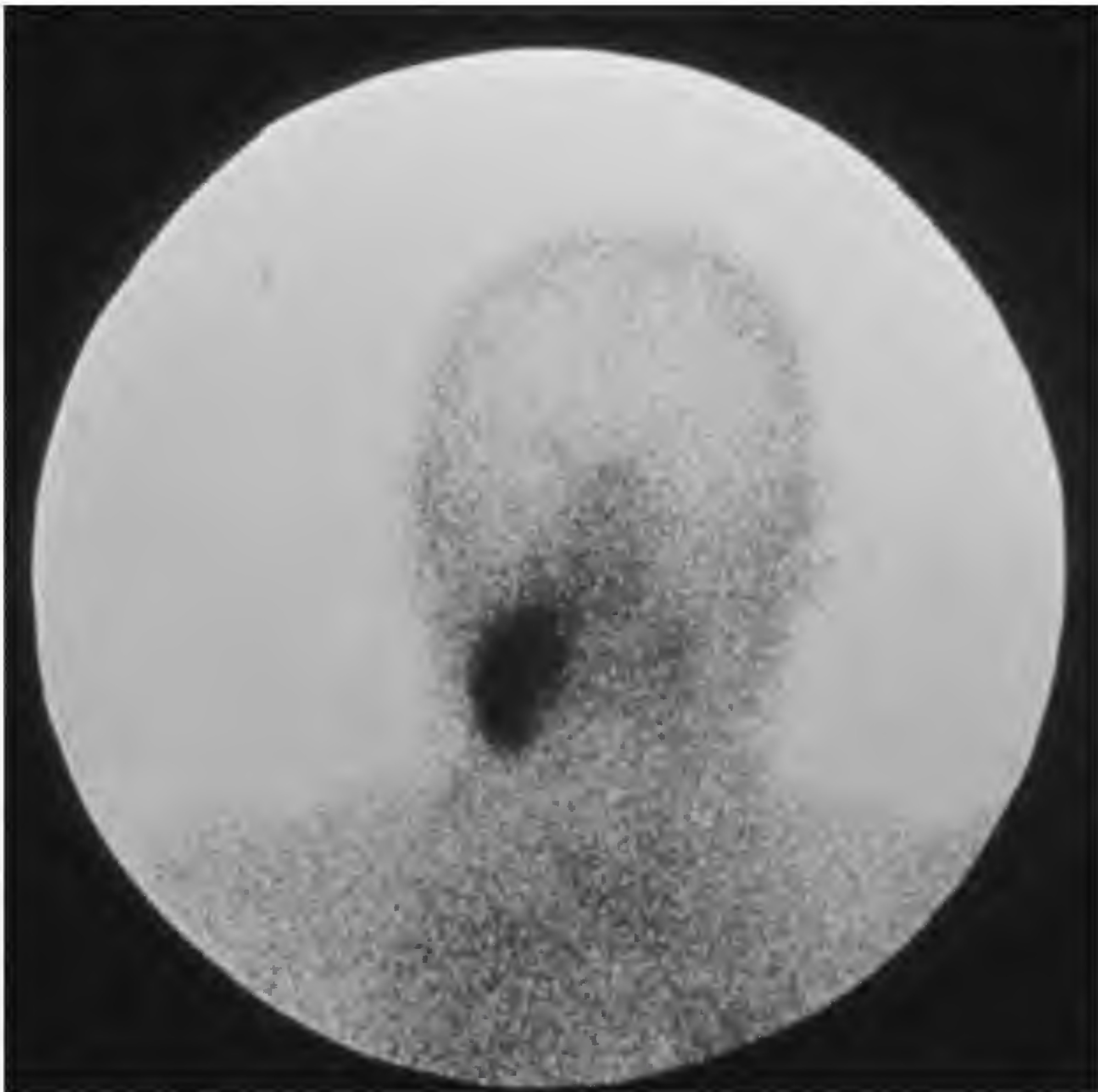
A. Conservative treatment.

B. Selective embolisation.

C. Radiation therapy.

D. Surgical excision.

E. Chemotherapy.



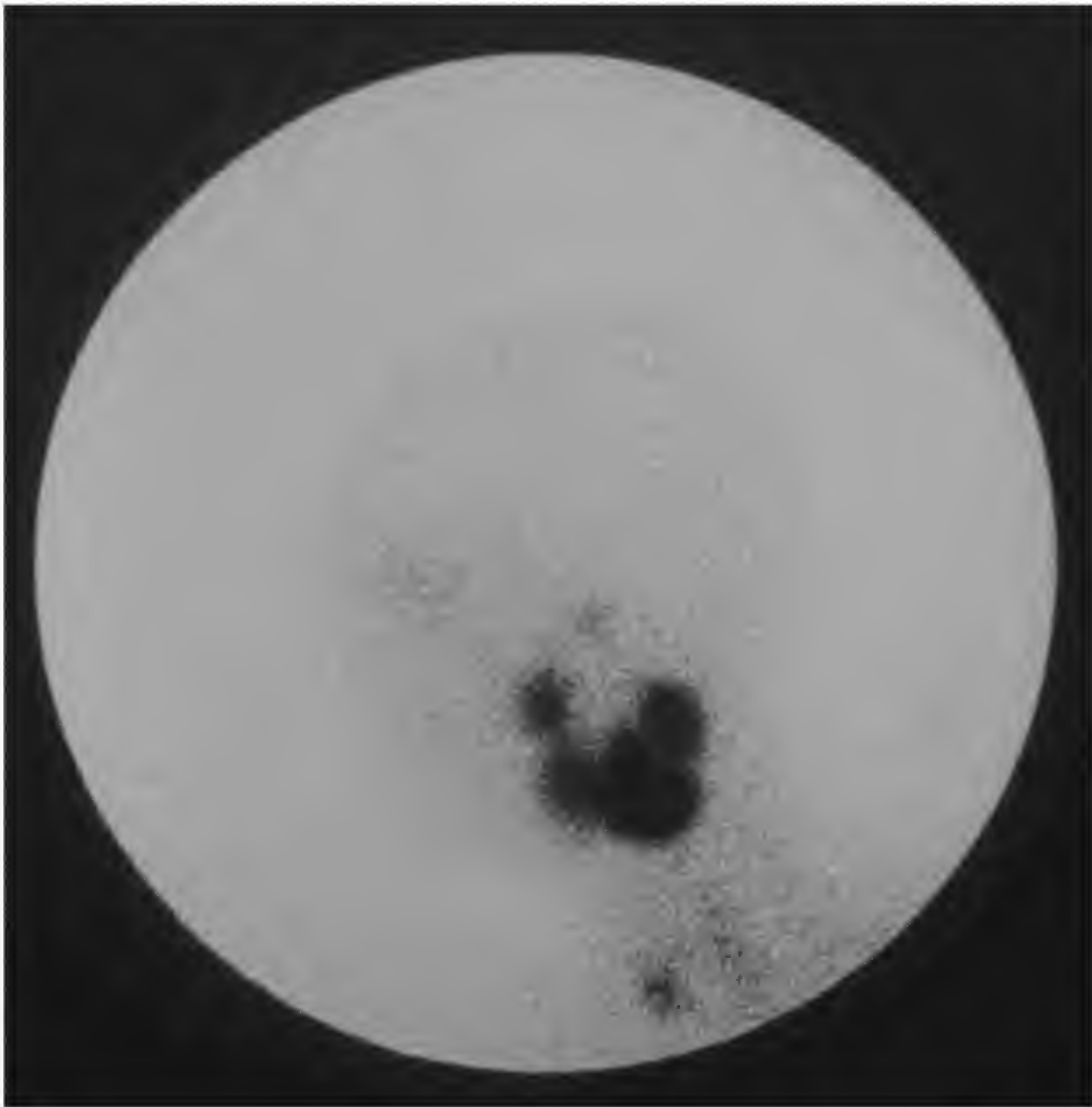


Fig. 30.3. a An OctreoScan of a carotid body tumour showing elevated uptake. **b** An OctreoScan with multiple tumours.



Fig. 30.4. The macroscopic anatomy of a carotid body tumour after surgical excision.

A surgical excision of the carotid body tumour was performed (Fig. 30.4). There were no complications, the patient recovered quickly and was discharged after 2 days. Pathological anatomical examination confirmed the diagnosis.

Question 4

What can be the complications due to surgical excision?

A. Deafness.

B. Horner's syndrome.

C. Vocal cord paralysis.

D. Paresis of the mandibular branch of the trigeminal nerve.

E. Ipsilateral tongue paresis.

Commentary

Paragangliomas are usually benign tumours derived from the paraganglia, a collection of neuroendocrine tissues which have a close relationship to the autonomic nervous system [1]. The normal paraganglia play an important role in homeostasis either by acting directly as chemical sensors or by secreting catecholamines in response to stress. The paragangliomas are generally divided into two groups, those occurring in the head and neck region and those occurring elsewhere, with the adrenal medulla being the most frequent site. In the head and neck, common locations of paragangliomas are the carotid bifurcation, the jugular foramen, the vagal nerve and the middle ear. Tumours in the carotid bifurcation, the carotid body tumours, are the most common type (60 percent) and are usually referred to the vascular surgeon. Another name commonly used for carotid body tumours is chemodectoma because carotid body tumours are tumours of the chemoreceptor system.

The carotid body is a highly vascularised, reddish-brown coloured, ellipsoid structure measuring $3 \times 5 \times 1.5$ mm and located in the adventitia of the bifurcation of the common carotid artery [2]. The carotid body has a homeostatic role and functions as an oxygen sensor. It stimulates the cardiopulmonary system in hypoxia through afferent input by way of the glossopharyngeal nerve to the medullary reticular formation [3].

Carotid body tumours can develop spontaneously and be induced by chronic hypoxia. The latter includes living at high altitudes and certain medical conditions (patients with chronic obstructive pulmonary disease, cyanotic heart diseases) [4–6]. Familial cases are reported in the literature in the percentage range 10–50 percent [7] but the range is probably 10–15 percent. They are frequently bilateral or multifocal and have an earlier age of detection. The inheritance pattern of paraganglioma is autosomal dominant modified by genomic imprinting. To date, four genetic loci have been identified [8–10]. The overwhelming majority of the tumours are benign but local expansion can cause cranial nerve deficits, invasion of the skull base, and eventually compression of the brain stem. Malignancy is mostly cited as 5 percent of cases [11], but in practice it is rarely seen. Malignancy cannot be defined on histology of the tumour itself but the unique criterion of malignancy is the presence of metastases [12], mostly in regional lymph nodes.

Clinical Presentation

Carotid body tumours have to be distinguished from enlarged lymph nodes, branchial cleft cysts, parotid tumours, thyroid tumours, neurogenic tumours and carotid artery aneurysms. A detailed history and physical examination will eliminate most other possibilities. Carotid body tumours can occur at any age, but they are typically diagnosed between the third and sixth decades of life [13]. The presence of a slowly growing, asymptomatic palpable mass in the anterior triangle of the neck must raise suspicion for this diagnosis (Fig. 30.1). In addition to the painless

mass, the patient may present with pain, hoarseness, dysphagia, Horner's syndrome, tongue paresis and vertigo. The tumours are generally non-functional; catecholamine secretion is present in only 5 percent of patients [11] and can cause hypertension. On physical examination, a carotid body tumour is noted to be pulsatile and can be moved laterally, but not vertically because of adherence to the carotid artery. A bruit may be auscultated over the mass, but this is a rare condition. The cranial nerves should also be evaluated. For instance, the tongue should be examined since paresis of the hypoglossal nerve can cause tongue dysfunction. Paresis of the vagal nerve can cause hoarseness as a result of vagal nerve paresis or paralysis. **[Q1: C]**

An echo-duplex is usually performed first, to differentiate between a carotid body tumour and other items on the differential diagnosis list. For further investigation, arteriography has long been regarded as the gold standard for diagnosis, but this is no longer the case. Magnetic resonance imaging (MRI) (Fig. 30.5) can reveal a circumscript mass at or above the carotid bifurcation. Use of contrast material usually shows a "salt and pepper" appearance caused by vessels with signal-void within the tumour stroma. Both MR angiography and digital subtraction angiography reveal marked vascularity of the tumour, and this may help to differentiate these hypervascular tumours from other tumours. If the tumour produces catecholamines, an MIBG (meta-iodobenzylguanidine) scan can be used or plasma levels can be checked.

An MIBG scan is a nuclear scan that uses an injected radioisotope to localise catecholamines; they are mostly used to locate or confirm a phaeochromocytoma. The patient receives an injection into a vein and imaging will take place between 1 and 4 days later. Somatostatin receptor scintigraphy has a much higher sensitivity for paraganglioma than an MIBG scan. Somatostatin receptor scintigraphy can be used to detect multiple paragangliomas because paragangliomas contain somatostatin-receptor carrying tissue (Fig. 30.3). If a carotid body tumour is suspected, a fine needle aspiration should **not** be performed and certainly an incision biopsy should be avoided in all cases. The diagnosis of a carotid body tumour is difficult to make on fine needle aspiration and this procedure can give rise to unnecessary complications such as massive bleeding. **[Q2: E, A, B, C, D]** It is of interest to note that more patients have died from the complications of invasive diagnostics and surgical treatment than from the natural cause of the disease. So the answer to Question 2 should be **E, A, B**. Answers **F** and **G** are obviously wrong and answers **C** and **D** are subjects of discussion.

Treatment

The preferred treatment for carotid body tumours is either conservative or surgical. Excision is the preferred definitive treatment, although the postoperative morbidity rate as quoted in the literature is rather high. Morbidity includes cranial nerve dysfunction, mostly of nerves X and XII. Tumour size is important; larger tumours have a higher incidence of complications [7]. **[Q4: A, B, C, D, E]** All the answers given in Question 4 are correct.

Postoperative mortality should not exceed 2–5 percent and occurs only in large tumours, while mortality is negligible in small tumours. Damage to the wall of the carotid artery, especially in the bifurcation, which is difficult to repair because the vessel wall is very thin as a result of dissection in the subadventitial space, may force

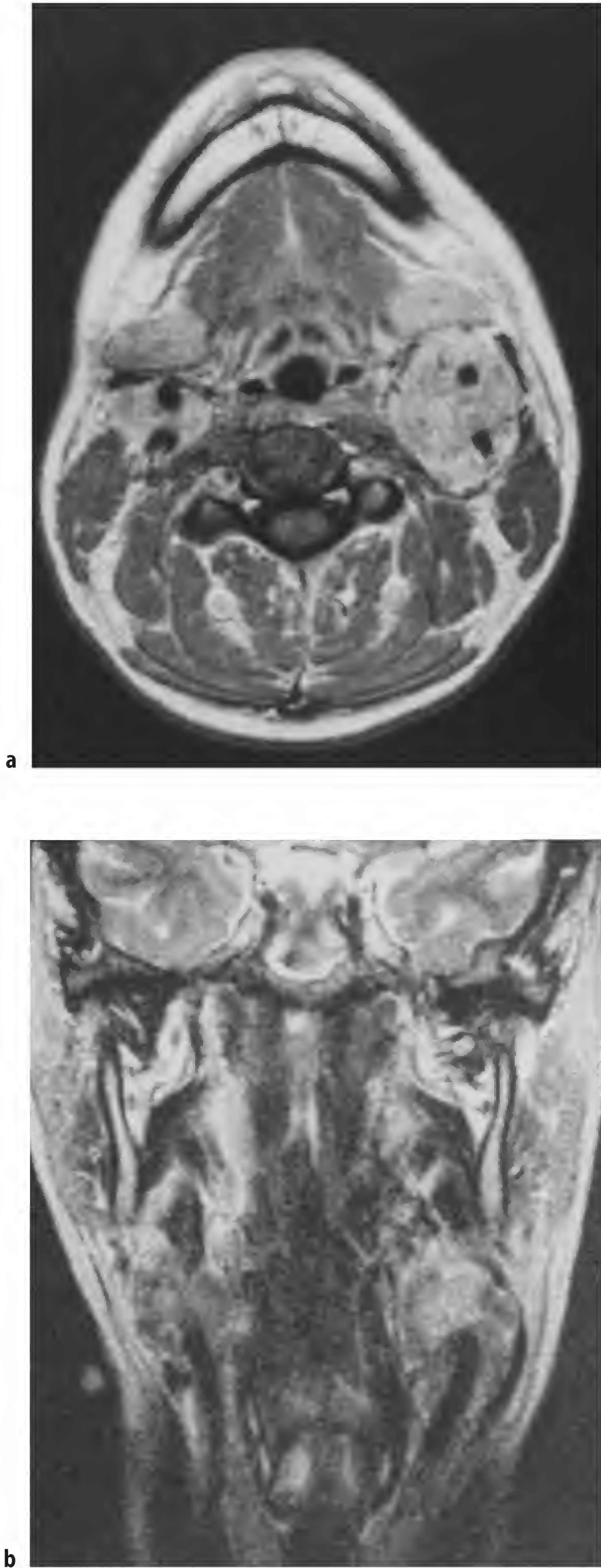


Fig. 30.5. a, b An MRI of a carotid body tumour.

the surgeon to clamp the internal carotid artery, sometimes leading to ischaemic stroke and death.

In 1972, Shamblin proposed a surgical classification for carotid body tumours based on their tendency to encase the carotid arteries. Shamblin group I are small tumours with minimal attachments to the carotid vessels; surgical excision can be performed without difficulty. Shamblin group II tumours are larger, and Shamblin group III are large tumours that encase the carotid arteries, which makes them more difficult to resect. Sometimes, it is even necessary to sacrifice the carotid bifurcation to be replaced by a venous or synthetic interposition graft in order to reconstruct the carotid artery [14].

Embolisation of the feeding branches of the external carotid artery can be performed a few days prior to surgery with the intention to decrease blood loss during operation. However, this is an area of continuing controversy. Some groups claim that embolisation decreases blood loss [15–17]; other groups have not found the embolisation procedure helpful and they warn of the increased risk of stroke caused by emboli to the brain through collateral pathways [18, 19]. Embolisation has been used in the past in very high-risk patients who probably would not tolerate surgical excision. It now seems to be largely abandoned because tumour growth appears to be stimulated in the non-necrotic hypoxic areas of the tumour. Radiation therapy and chemotherapy are seldom used as a treatment for carotid body tumours. Radiotherapy might be an alternative to surgery. It may be the treatment option for large, fast growing tumours, which are not eligible for surgery, and it may be effective in arresting growth. However, radiotherapy does not result in complete eradication. There is no evidence and there are no prospective trials showing that chemotherapy might be effective against carotid body tumours. **[Q3: A, D]**

Summary

Paragangliomas are slowly growing benign tumours. In the head and neck region, the carotid body tumour is the most common type. The diagnosis is suspected from the patient's history and physical examination. A somatostatin receptor scintigraphy is a reliable method for confirming the diagnosis and detecting multiple tumours at the same time. If the carotid body tumour is small and there is no documented growth, a wait-and-see policy is justified. A fast growing or large tumour should be treated surgically, cranial nerve dysfunction being the most common postoperative complication.

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31. Vertebrobasilar Ischemia: Embolic and Low-flow Mechanisms

Ramon Berguer

Vertebrobasilar Ischemia: Embolic Mechanism

A 51-year-old male experienced over a period of 6 months a major stroke and several transient ischemic attacks (TIAs) of vertebrobasilar distribution. The original episode consisted of loss of balance, loss of coordination, and loss of the left visual field while driving a bus, which resulted in a road accident. Since then, he had experienced four additional episodes of aphasia and paraparesis lasting for 4–5 h. A diagnosis of vertebral artery dissection was made at the local hospital and he was placed on Coumadin. Concomitant diagnoses were hypertension, non-insulin-dependent diabetes, and hypercholesterolemia. In spite of adequate international normalized ratio (INR) levels, his symptoms continued and he was referred to us.

On admission, magnetic resonance imaging (MRI) showed right occipital and left cerebellar infarctions (Fig. 31.1).

Question 1

The work-up of this patient presenting with symptoms of vertebrobasilar ischemia and MR evidence of infarction in the posterior region must include:

A. CT scan of the brain.

B. Carotid-vertebral duplex.

C. Electroencephalogram (EEG).

D. Arteriogram.

E. Echocardiogram.

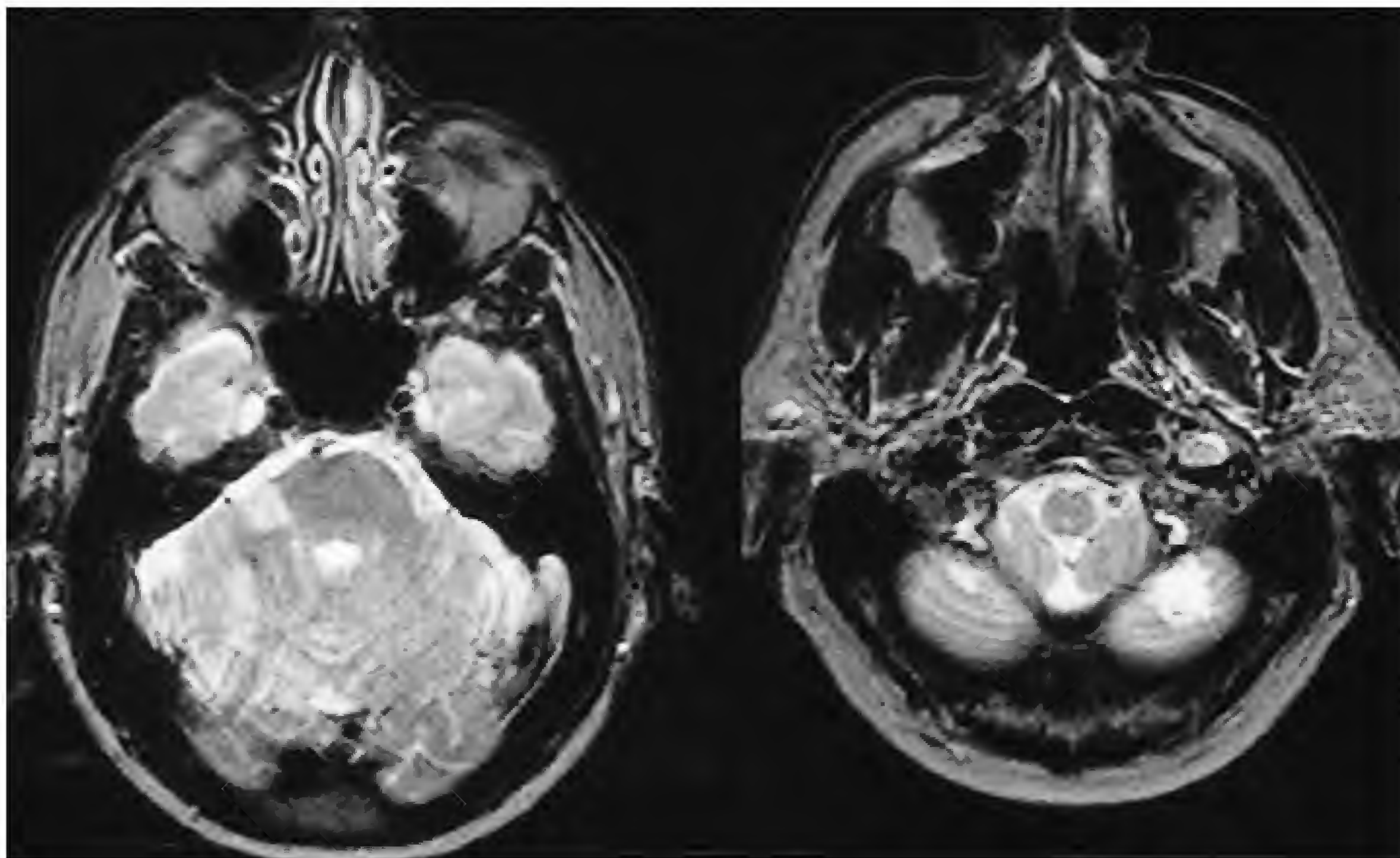


Fig. 31.1. MRI showing cerebellar and brainstem infarctions.

Question 2

The etiology of infarction in the posterior circulation territory is:

A. Distal embolization of atheromatous material from vertebral or basilar artery lesions.

B. Arrhythmia.

C. Bilateral carotid disease in patients with absent vertebral arteries.

D. Traumatic or spontaneous dissection of the vertebral artery.

E. Transient drop in central aortic pressure in a patient with severe bilateral stenoses of both vertebral arteries.

An arteriogram showed a 60 percent stenosis in the fourth portion of the right vertebral artery, and a tenuous, incomplete (dissected) left vertebral artery, which, at the level of C1, became a normal artery and, higher up, joined with the opposite vertebral artery (Fig. 31.2). A diagnosis of embolizing dissection of the left vertebral artery was made. Because the dissection was not responsive to medical therapy, the patient underwent a bypass from the left internal carotid to the left (suboccipital) vertebral artery using a saphenous vein [1]. The proximal vertebral site of the embolizing dissection was ligated above C1, immediately below the distal anastomosis of the carotid-vertebral bypass (Fig. 31.3). The patient did well from this operation and stopped having symptoms. His anticoagulation was discontinued. He remains asymptomatic after 5 years of follow-up.

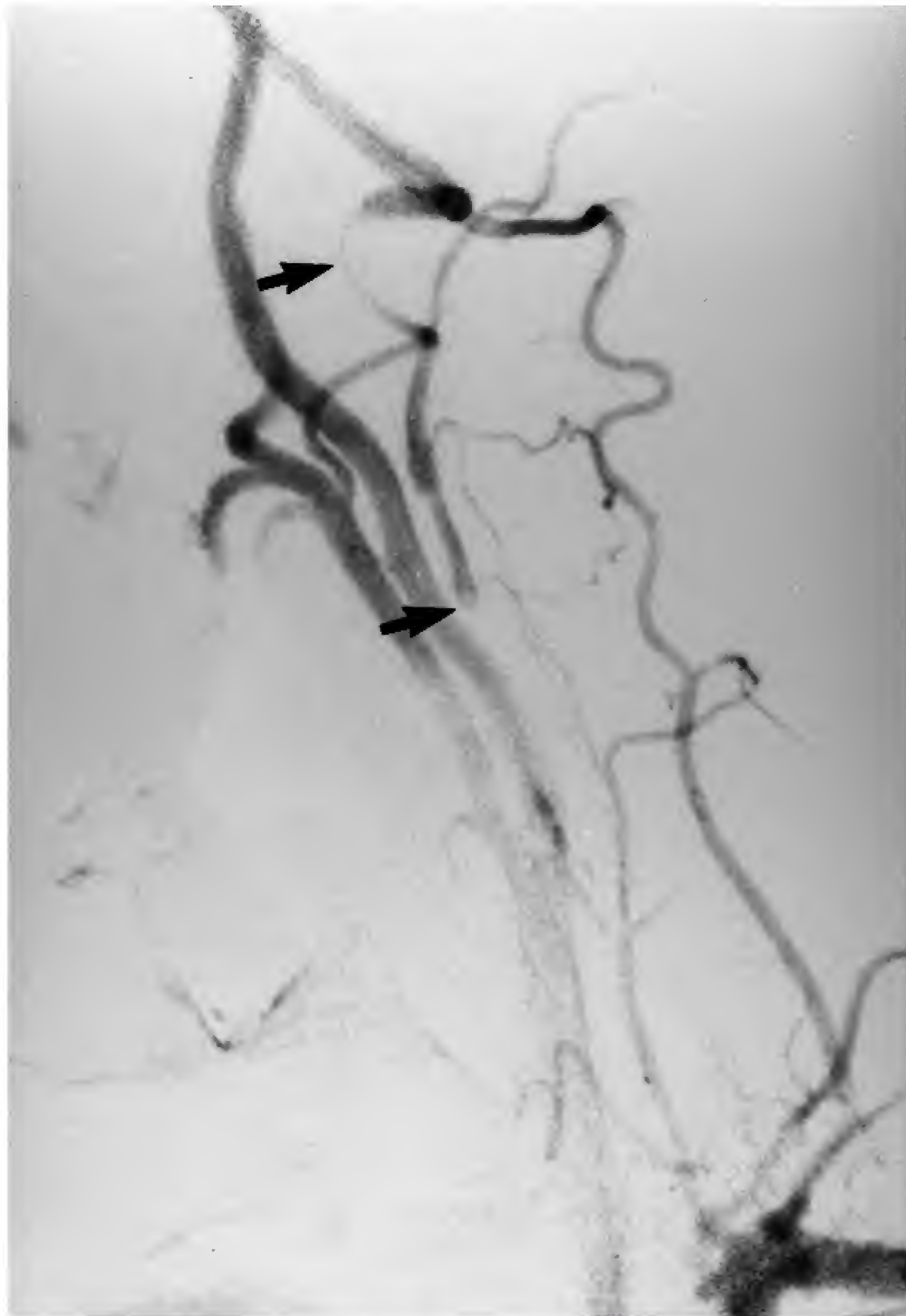


Fig. 31.2. Arteriogram: dissection of the left vertebral artery, which is occluded from its origin to C4 (lower arrow), dissected and partially occluded from C4 to C1 (between arrows), and normal distal to C1.

Question 3

Once the objective diagnosis of vertebral artery dissection is made in a patient with vertebrobasilar symptoms the next step is:

A. Anticoagulation with heparin, then Coumadin.



Fig. 31.3. Postoperative carotid arteriogram showing a saphenous vein bypass from the distal cervical internal carotid to the vertebral artery beyond C1.

B. Stenting of the dissection followed by antiplatelet therapy.

C. Surgical bypass of the dissected segment with ligation of the proximal vertebral artery.

Commentary

Dissection of the vertebral artery may occur spontaneously or result from trauma [2–6]. The traumatic event is usually an exaggerated extension or rotation of the neck as may occur during sports and deceleration injuries.

Clinical presentation of dissection of the vertebral artery starts with pain over the posterolateral aspect of the neck irradiating to the nuchal area. There may be an interval of several days between the initial pain, announcing the dissection, and the development of clinical symptoms. The latter are ischemic manifestations of the dissection and appear in 60–90 percent of patients after an interval of several days, usually 1–2 weeks. A carotid-vertebral duplex would not provide a discriminating datum to help in the decision on the management of our patient because: (i) it would be only confirmatory for a possible concomitant carotid atheroma, which has never been shown to be the source of infarction in the cerebellum or brain stem; (ii) infarction in the posterior region can only be evaluated by means of an arteriogram. The latter will provide in addition the information about the carotid arteries that you would have derived from the carotid-vertebral duplex. **[Q1: D, E] [Q2: A, D]**

The treatment of symptomatic vertebral artery dissection is empirical with systemic anticoagulation. Patients with posterior fossa symptoms should undergo MRI before starting anticoagulation to rule out a subarachnoid hemorrhage. The latter may occur following dissection and rupture of the fourth (intracranial) segment of the vertebral artery.

Anticoagulation is empirically used for the treatment of symptomatic dissection because the ischemia that follows is the consequence of embolization from the double channel, not a low-flow effect. The fear of distal extension of the dissection with anticoagulants has prompted some leading experts to give antiplatelet therapy to patients with local symptoms (pain) and evidence of dissection but without central manifestations of ischemia (central nervous system deficits or MR evidence of infarction). Patients with massive infarction are not anticoagulated to avoid intraparenchymal bleeding. There is no indication for wire-catheter-stent manipulation of a dissected vertebral. In patients who are anticoagulated appropriately and continue to have intermittent symptoms, the dissected vertebral artery is considered to be the source of emboli. **[Q3: A]** In these circumstances, and if technically feasible, the dissected segment is excluded and bypassed [7, 8].

Vertebrobasilar Ischemia: Low-flow Mechanism

A 62-year-old woman with a healthy lifestyle presented with a history of dimming of the visual field and passing out when she turned her head to the extreme right. Three months before, she had been evaluated elsewhere with a history suggestive of amaurosis fugax and bouts of imbalance and vertigo when she turned her head to the right. A carotid endarterectomy had been performed at another institution.

She continued to have severe vertebrobasilar symptoms with head turning. She had a myocardial revascularization 20 years ago, at which point she stopped smoking.

On examination, the patient appeared healthy, with normal and equal (124/80 mm Hg) blood pressure in both brachial arteries. Neurological examina-

tion under resting conditions was normal. Her neck was silent. When her head was turned to the right, the patient developed dimming of vision, loss of balance, and a sensation of passing out. The arteriogram available from the previous operation carried out elsewhere showed a clearly dominant large left vertebral artery, but we could not see clearly the distal segment of the vessel. The right vertebral artery was small and diseased severely to a preocclusive level throughout its second segment. There was no evidence of posterior communicating arteries.

Because the symptoms were repetitive and induced posturally, the patient was scheduled for a dynamic arteriogram. First, we obtained a view with a selective subclavian injection of the dominant left vertebral in the neutral position, which was normal. Following this, the patient's head was turned to the right; when she became symptomatic, the contrast injection was repeated (Fig. 31.4). This revealed a severe compression of the vertebral artery at the level of the pars atlantica of C1.

The patient underwent exploration of the suboccipital space with dissection and exposure of the vertebral artery where it crossed the lamina of C1. The compression mechanism was between the sharp upper edge of the lamina and the occipital bone. A laminectomy was carried out to provide space for the artery to pass from the exit of the transverse foramen of C1 to the foramen magnum without bony compression (Fig. 31.5). The artery was examined by palpation and direct duplex interrogation; we could find no element of plaque or stenosis in the lumen once the artery was freed and the laminectomy completed. The patient became asymptomatic. Full-range motion of the neck no longer caused syncope or vertigo.

Question 1

Which of the following statements regarding posturally induced symptoms is true?

A. The mechanism for ischemia is the restriction of flow by external compression of the artery.

B. The mechanism for ischemia is embolization from the damaged wall (dissection) or thrombus overlying the endothelial lining of the artery at the site of trauma.

C. Both mechanisms may exist.

Question 2

Which of the following statements are correct?

A. When dynamic symptomatic compression of the vertebral artery is demonstrated, angioplasty (with or without stent) is never indicated.

B. Angioplasty of a stenosed or dissected vertebral artery at the suboccipital level is likely to result in rupture of the artery or formation of an arteriovenous fistula.

C. Angioplasty and stenting of the distal vertebral artery is successful in stenosing lesions caused by external compression.



Fig. 31.4. Selective injection of a left subclavian artery while the patient is experiencing symptoms with her head turned to the right. The single, dominant vertebral artery is severely compressed above C1 in its pars atlantica.

Commentary

In patients with low-flow ischemia secondary to extrinsic compression of the artery, the clinical picture is repetitive and can be induced by manipulating the patient's head in the trigger position.

Patients with symptoms occurring with head rotation or extension should have a dynamic arteriogram to show the anatomic lesion (extrinsic compression) at the



Fig. 31.5. Three-dimensional reconstruction of a CT scan of the craniocervical junction. The lamina of C1 has been removed.

same time as the patient experiences symptoms. Patients with low-flow symptoms (repetitive) and no evidence of embolization (negative MRI) may show deformity/compression of one vertebral artery but a normal contralateral vertebral artery during head rotation or extension. If the contralateral, undisturbed artery is of normal size and empties normally into the basilar artery, then the role of the compression of one vertebral artery causing the symptoms must be doubted.

The suboccipital approach permits access to the vertebral artery from the transverse process of C2 to the foramen magnum. The techniques used to relieve compression at the suboccipital level are laminectomy, laminectomy plus bypass, and bypass alone.

Vertebrobasilar ischemia of postural origin is generally the consequence of mechanical compression of the vertebral artery by osteophytes (and occasionally ligaments) in its extracranial trajectory. The mechanism for symptoms is generally low flow in a dominant vertebral artery that cannot be compensated for by flow from a contralateral hypoplastic or absent vertebral artery. This compression is seen very rarely in the first segment (origin–C6) caused by compression by the tendon of the longus colli. It is usually observed in the second and third segments of the artery. In the second segment (C6–C2), the artery is usually compressed by osteophytes, and the symptoms generally appear with rotation of the neck. In the third segment (C2–C0), the compression may occur at C1 or, more commonly, in the pars atlantica of the artery between C1 and the foramen magnum. The artery is

compressed between the sharp upper edge of the lamina of C1 and the occipital ridge. **[Q1: C]**

The ischemic symptoms are usually the consequences of low flow through a dominant vertebral artery because of complete or near-complete occlusion at the latter by an osteophyte. Less frequently, the ischemic effects may be embolic from the mural thrombi that develop at the site of repetitive trauma on the artery by the offending osteophyte. In other cases, the artery may dissect at the point of repetitive traumatic compression, which results in its occlusion and/or distal embolization.

Symptoms in patients with vertebrobasilar ischemia from the low-flow mechanism are repetitive and can be reproduced every time the neck is brought to the trigger position. Patients with vertebrobasilar ischemia of embolic origin usually present with a clinical stroke or TIA in different areas. MRI in the low-flow group is usually normal, but in the embolic group it will show cerebellar, brainstem or occipital infarctions.

An arteriogram is needed to outline precisely the point of compression and to discern the possibility of a dissection and/or tandem lesions. It is also important to outline the entire course of the opposite vertebral artery to establish whether it is complete, normal or hypoplastic, and whether at the time of the provocative dynamic arteriogram the opposite vertebral artery fills the basilar artery normally while the patient has symptoms. The latter suggests that the mechanism of symptoms is not low flow.

There is no role for angioplasty, with or without stent, in the treatment of extrinsic compression of the vertebral artery. Balloon dilation of the thin-walled vertebral artery against the hard bony prominence of an osteophyte is likely to result in the rupture of the arterial wall and the formation of a false aneurysm or an arteriovenous fistula.

If the compression of the vertebral artery is limited to the V2 segment (C6–C2), then the single or multiple elements of compression are bypassed by reconstructing the artery to the level of C1. This is done through an anterior approach [8]. In dynamic compression at the suboccipital level, the approach is posterior [1] and the treatment consists of a laminectomy, a bypass or both. In the case of a bypass, the inflow is obtained from the high cervical carotid. The latter is exposed by moving aside the cranial nerves that block access to the internal carotid when approached posteriorly. **[Q2: A, B]**

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32. Neurogenic Thoracic Outlet Syndrome

Richard J. Sanders

A 30-year-old woman presented with complaints of pain in her neck, right shoulder, right trapezius, right anterior chest wall, right arm, elbow, and forearm; occipital headaches every other day; numbness and tingling in all fingers of the right hand, worse in the fourth and fifth fingers; aggravation of her symptoms when elevating her arms, especially to comb or blow dry her hair or drive a car; weakness of her right hand and dropping coffee cups; and coldness and color changes in her right hand. The symptoms had been present for one year and began following a rear-end collision.

Her history began one year ago when her automobile was sitting still at a traffic light and another vehicle hit her from the rear. She wore a seat belt and recalled going forward and backward, but did not recall what happened to her neck at the time of the accident. She had no immediate symptoms. On the next day she awoke with a sore neck and pain above her shoulder blades. A few days later, she began noticing headaches in the back of her head that radiated forward to behind her eyes, and the neck soreness became progressively painful. Two or three weeks later, pain developed in the right shoulder area and down the right arm. Several weeks later, numbness and tingling developed in the fingers of the right hand, more noticeable in the ring and baby fingers. Because of severe, persistent right shoulder pain, arthroscopic repair of the right shoulder had been performed 6 months ago with partial improvement of her shoulder pain, but no change in any of her other symptoms.

Her occupation was a legal secretary. Since the accident, although she had been able to return to work, she was now able to work only 4 hours a day. She could not type for more than 10 minutes because the pain and numbness in her right hand was too uncomfortable. At home she could do light housework only. She could not vacuum, wash windows or floors, or lift heavy laundry baskets.

Diagnostic studies to date included cervical spine X-rays, which were normal, and an electromyography/nerve conduction velocity (EMG/NCV) study, which revealed very mild nonspecific changes in the ulnar nerve distribution, but was close to normal.

Treatment to date included 6 months of physical therapy with the following modalities: heat, massage, ultrasound, neck stretching exercises, and posture

correction. She was continuing neck stretching exercises at home on a daily basis emphasizing doing each stretch slowly, holding each stretch for a minimum of 15 seconds, and performing no more than three repeats at each session. In spite of this treatment, there was no improvement in her symptoms.

Question 1

What is the most common cause of neurogenic thoracic outlet syndrome (TOS)?

A. Neck trauma.

B. Cervical rib.

C. Anomalous bands.

D. Abnormal first rib.

E. All of the above.

On physical examination there was supraclavicular tenderness over the right scalene muscles but no tenderness over the left scalenes; a positive Tinel's sign over the right brachial plexus and a negative sign over the left; and reproduction of arm and hand symptoms with pressure over the right scalene muscles, but no such symptoms with pressure over the left scalene muscles. Head rotation and head tilting each caused pain in the contralateral hand and arm when turning and tilting to the left side. This did not occur when rotating and tilting to the left side. Abducting the arms to 90° in external rotation (AER position) reproduced the right arm and hand symptoms within 15 seconds while no symptoms developed on the left side.

Scalene muscle block, injecting 4 ml of 1% lidocaine into the right anterior scalene muscle area, resulted in significant improvement in most of her physical findings.

Question 2

The diagnostic criteria for neurogenic TOS include which of the following?

A. History of neck trauma.

B. Paresthesia in the hand involving all five fingers, more frequently in the fourth and fifth.

C. Pain in the neck, shoulder, and upper extremity.

D. Occipital headaches.

E. Scalene muscle tenderness and duplication of symptoms in the 90° AER position.

F. Cut-off of the radial pulse on Adson's or 90° AER positioning.

G. Positive response to the scalene muscle block.

Question 3

Which of the following conditions can coexist with TOS or require differentiation from it?

A. Carpal tunnel syndrome.

B. Biceps/rotator cuff tendinitis or impingement syndrome.

C. Cervical spine disease-disc, arthritis, spinal stenosis, cervical spine strain, etc.

D. Ulnar nerve entrapment at the elbow (cubital tunnel syndrome).

E. Fibromyalgia.

F. Brachial plexus injury.

G. Brain tumor.

Question 4

The indications for surgical decompression of the thoracic outlet areas are:

A. Failure of conservative treatment after a trial of several months.

B. All other associated conditions have been recognized and treated as completely as possible.

C. Symptoms are interfering with work, sleep, recreation, or activities of daily living.

D. All of the above.

Because of persistent symptoms in spite of adequate conservative therapy, and because she was partially disabled at work and at home, a supraclavicular anterior and middle scalenectomy, brachial plexus neurolysis, and first rib resection were performed.

Question 5

Which surgical procedures are acceptable to decompress the thoracic outlet area?

A. Transaxillary first rib resection.

B. Supraclavicular anterior and middle scalenectomy with brachial plexus neurolysis.

C. Supraclavicular anterior scalenectomy with or without brachial plexus neurolysis.

D. Supraclavicular anterior and middle scalenectomy, first rib resection, and brachial plexus neurolysis.

E. All of the above.

Question 6

What are the major complications of TOS surgery?

A. Brachial plexus traction injury.

B. Phrenic nerve injury.

C. Subclavian artery injury.

D. Subclavian vein injury.

E. Long thoracic nerve injury.

F. Second intercostal brachial cutaneous nerve injury (transaxillary approach only).

G. Thoracic duct injury (left side, supraclavicular approach only).

H. Supraclavicular nerve injury (supraclavicular approach only).

I. Horner's syndrome (supraclavicular approach only).

J. All of the above.

She tolerated surgery well, had no postoperative complications, and was discharged from the hospital on the second postoperative day. After 4 weeks of convalescence at home, she returned to work, 4 hours a day. After one month she was able to resume her job on a full-time basis. While most of her symptoms had improved, she still noticed occasional paresthesia in her hand and pain in her right shoulder when working for long periods. Her headaches were completely gone. She was pleased with her improvement from surgery even though she was not back to normal.

Question 7

What are the long-term results of surgical decompression of the thoracic outlet area?

A. 90% success.

B. 75% success.

C. 60% success.

D. 40% success.

E. None of the above.

Commentary

There are three types of thoracic outlet syndrome (TOS): arterial, venous, and neurogenic. Neurogenic TOS comprises more than 95 percent of all TOS cases and is

the most difficult to diagnose and treat. The etiology of neurogenic TOS in most patients is either a hyperextension neck injury or repetitive stress at work. The mechanism of neck injury from repetitive stress is a little obscure, but it probably comes from the worker's hands being occupied in one place so that the worker is constantly rotating his/her neck back and forth to perform the job or talk to people. Holding a telephone between ear and shoulder while typing is also a common form of neck strain. While some TOS patients have cervical ribs or congenital cervical bands, these are regarded as predisposing conditions and seldom are the primary cause. These patients usually do not develop symptoms until they experience some form of neck trauma.

Although first rib resection has become a standard form of therapy for neurogenic TOS, the first rib is rarely the cause of the symptoms. The pathology is tightness and scarring of the scalene muscles [1]. Rib resection is successful because the anterior and middle scalene muscles must be divided in order to remove the rib. Thus, by necessity, first rib resection includes scalenotomy and it is probably the latter that relieves the symptoms. **[Q1: A]**

The diagnosis of neurogenic TOS is by history and physical examination. This is not a diagnosis of exclusion. The typical history includes some type of neck trauma, although the patient does not always remember the incident, especially if there was no litigation involved. It is the job of the examiner to thoroughly ask about neck trauma. The symptoms usually include pain, paresthesia, and weakness in the upper extremity, but over 75 percent of patients also complain of neck pain and occipital headaches. The latter symptoms are not the result of brachial plexus compression; rather, they result from stretch injuries to the scalene muscles and referred pain to the back of the head. Most commonly paresthesia involves all five fingers of the hand, although it tends to involve the ulnar side of the hand and forearm more often than the radial side. The significant physical findings are scalene muscle tenderness, a positive Tinel's and positive Spurling's sign over the scalene muscles, and duplication of symptoms with the arms in the 90° AER position. A cut-off of the radial pulse in either the Adson's or 90° AER position is not a reliable sign in establishing a diagnosis. Up to 60% of normal people cut off their pulses in these dynamic positions while most TOS patients do not cut off their pulses [2]. Not every patient will exhibit all of these criteria, but a diagnosis can be established if the majority of these criteria have been met [3] **[Q2: A, B, C, D, E, G]**

All symptoms of TOS are nonspecific. Other conditions that also exhibit similar symptoms include abnormalities of the shoulder, elbow, wrist, and parascapular muscles. It is quite common for TOS to coexist with some of these other conditions. **[Q3: A, B, C, D, E, F]**

In less than 1 percent of patients with neurogenic TOS, atrophy of hand muscles supplied by the ulnar nerve exists. In these patients, EMG studies demonstrate typical findings of ulnar neuropathy [4]. Otherwise, EMG and NCV studies are either normal or reveal nonspecific changes. Unfortunately, once atrophy develops, it is usually nonreversible [5]. At this stage, surgery can relieve pain and paresthesia, but not weakness.

Conservative therapy is always indicated first and is effective in the majority of patients [6]. Surgery should be regarded as a last resort. There are a variety of modalities of therapy for TOS patients, the most important being home exercises, including neck stretching, abdominal breathing, and posture correction. After being instructed by a physical therapist the patient carries out the program on a daily basis at home. Hands-on therapy by a physical therapist is indicated for some of the

associated diagnoses that coexist with TOS. Because neck traction, weights, resistance exercises, and strengthening exercises tend to make TOS symptoms worse, we do not recommend them for TOS patients.

Some patients are refractory to all forms of physical therapy. If there is no improvement after several months of exercises, the patient's options are to either live with the symptoms or consider surgical decompression of the thoracic outlet. To be a candidate for surgery, in addition to failing conservative therapy after a trial of several months, the patient should have had all associated diagnoses treated and the symptoms should be partially or totally disabling. **[Q4: D]**

That there is more than one acceptable surgical procedure from which to choose indicates that no one operation has proved itself to be greatly superior to any other. In 1972, after performing transaxillary first rib resection [7] for several years, we were disappointed to find the long-term success rate was under 70%. We then changed to supraclavicular anterior and middle scalenectomy with brachial plexus neurolysis but were again disappointed to discover the success rate was identical to transaxillary first rib resection. The next choice of procedure was supraclavicular anterior and middle scalenectomy plus first rib resection through the same supraclavicular incision [8, 9]. With this combined operation our early results were a few percentage points better than the first two operations, but the difference was not statistically significant. Other observers who have compared scalenectomy alone to scalenectomy with first rib resection have also not noted statistically significant differences between the two [10–12]. Finally, some surgeons still perform just anterior scalenectomy with neurolysis and report results that are similar to the more extensive procedures.[13, 14]. **[Q5: E]**

Major complications occur from all operations to decompress the thoracic outlet area regardless of the surgical approach. Injury to the subclavian artery and vein, brachial plexus, phrenic nerve, and long thoracic nerve are the most common serious complications. Less common are injuries to the thoracic duct and cervical sympathetic chain. Injuries to cutaneous nerves from either transaxillary or supraclavicular approaches are common. Plexus injury occurs from excessive traction, which at the time may not seem excessive. Plexus injury can also occur when a clamp on the subclavian artery to control bleeding accidentally includes a nerve of the plexus.

Plexus injury makes symptoms worse in 1 percent of patients. The incidence of temporary phrenic nerve injury during supraclavicular approaches is 6–10 percent because the phrenic nerve is often in the middle of the field and is very sensitive to even mild retraction [12]. **[Q6: J]**

The results of surgery are about the same for all procedures. The biggest variable is etiology. When the etiology is an auto accident related injury, the 1-year success rate is 75–80 percent; when the etiology is repetitive stress at work or a work injury, the success rate is 15 percent lower [11, 12, 15]. **[Q7: B** (for auto accident etiology), **C** (for work related and repetitive stress etiology)**]**

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33. Thoracoscopic Sympathectomy

Samuel S. Ahn, Huck A. Mandel and Kyung M. Ro

A 22-year-old female undergraduate student who had had bilateral palmar hyperhydrosis since the fifth grade presented with hyperhydrosis that was refractory to noninvasive treatment. Her sweating was severe, to the point that it dripped and was thus incapacitating. Her symptoms become more severe primarily when she was anxious or upset, but they also increased with physical activity or elevated temperatures. She was unable to shake or hold hands in social settings. She had difficulty taking exams because the sweat made the paper wet. She was currently looking for a job and felt socially inhibited when being interviewed. Conservative therapy had failed, trying Drysol, Robinul, Drionic and roll-ons. Her hyperhydrosis had also proven refractory to hypnotherapy, biofeedback and iontophoresis. She denied any axillary hyperhydrosis. Her past medical history was negative for any major illnesses, and she denied any thyroid conditions, hypertension or diabetes. She had had no previous lung infections or prior surgeries. Her family history revealed a sister and a maternal grandfather also positive for hyperhydrosis.

A review of symptoms was negative for weight loss, malaise, fatigue, chills and fever. The patient denied any constipation, diarrhoea, chest pain, shortness of breath or palpitations. She had no polydipsia, polyuria, or loss of strength or function in the extremities. She also denied any Raynaud's syndrome-like symptoms.

On physical examination, both hands were cool and clammy to the touch and exhibited severe dripping. Lung sounds were clear bilaterally. The neck was supple, with full carotid pulses and no thyromegaly or lymphadenopathy. No thoracic outlet compression was evident on examination.

In this patient with severe bilateral hyperhydrosis refractory to medical treatment, the plan was to proceed with bilateral thoracoscopic sympathectomy.

Question 1

What is primary hyperhydrosis, and what is its incidence?

Question 2

What is the natural history of primary palmar hyperhydrosis?

Question 3

What are the indications for thoracoscopic sympathectomy?

Question 4

What are the relative contraindications for the procedure?

The patient was taken to the operating room and intubated with a double-lumen endotracheal tube. She was then placed in the lateral decubitus position, with the ipsilateral arm abducted in the arm holder at a 90-degree angle. Strong radial pulses were palpated. The chest wall was prepped and draped in sterile fashion. Entrance into the chest was through three separate 7-mm ports. Soft flexible trocars were used. The first port was placed in the sixth intercostal space in the posterior axillary line, a second port in the sixth intercostal space in the mid-axillary line, and a third port in the third intercostal space along the anterior axillary line.

The ipsilateral lung was deflated mechanically and retracted anteriorly and inferiorly. The superior aspect of the sympathetic chain and the extent of surgical dissection were identified through visualisation of the subclavian artery. The azygous, innominate and subclavian veins, along with the phrenic and vagus nerves, were identified and preserved. Electrocautery was used solely to unroof the parietal pleura from rib segments 1–4, thereby exposing the sympathetic chain and ganglia. Electrocautery was not to be used in proximity to the nerve fibres. The rami communicantes of the second and third ganglion were cut (the fourth ganglion as well if the axillary hyperhydrosis is significant) while taking extreme precautions to avoid injury to the intercostal artery and vein. The dissection proceeded in a rostral fashion and was carried to the lower portion of the stellate ganglion. Next, the nerve of Kuntz was identified (this is a large branch of the T1 ramus that runs parallel and lateral to the trunk of the sympathetic chain at the inferior aspect of the superior stellate ganglion). The sympathetic chain was resected inferiorly and caudally to the stellate ganglion at the level of the superior margin of the second rib. The third ganglion and the chain just caudal to the end of the third ganglion were subsequently divided, corresponding to the level of superior aspect of the fourth rib. The chain ganglion was sent to pathology for intraoperative histological confirmation. A 16 Fr chest tube was inserted through the middle port and placed under direct visualisation. The lung was then reinflated using a positive pressure of 30–40 mm Hg, and adequate inflation was confirmed by endoscope. The port was closed using 3-O Dexon running sutures. The patient was then repositioned on the contralateral side and completely reprepped and redraped, and the procedure was repeated on the other side.

Question 5

What are the short-term complications associated with the procedure?

Question 6

What are the long-term complications associated with the procedure?

Immediately following the operation, a chest X-ray was performed to rule out pneumothorax or pleural effusions. No air leaks were present, and the chest tubes were removed in the recovery room. The patient was placed on oral analgesics and discharged from the hospital the same day.

On her 1-week postoperative visit, the patient had no evidence of Horner's syndrome, compensatory hyperhydrosis, neuralgia or bradycardia. The patient had complete resolution of her palmar hyperhydrosis and no recurrence of symptoms at 2-years' follow-up.

Question 7

What are the long-term results of thoracoscopic sympathectomy?

Question 8

What are the results of the thoracoscopic sympathectomy procedure for palmar hyperhydrosis in children and adolescents?

Commentary

Primary hyperhydrosis is a pathological condition of overperspiration caused by oversecretion of the eccrine sweat glands [1]. The aetiology is unknown. An exaggerated response to emotional stimuli, heat and physical effort has been implicated [2, 3]. It has also been suggested that hyperhydrosis is a condition of central origin, with resultant excessive stimulation of the sweat glands by an overactive sympathetic system; it may also be attributed to an autonomic dysfunction [4, 5]. Primary hyperhydrosis may be confined to the palms in some patients; however, the axillae and soles are also often affected.

The incidence of primary hyperhydrosis is poorly understood. The only epidemiological study was conducted by Adar et al. [6], who reported an incidence of 0.6–1% with no evidence to suggest a greater male or female predominance. Over the past several years, various investigators have described positive family histories, ranging from five to 50 per cent, with 40 per cent of these patients having excessive sweating [7–12]. Although many ethnic predispositions have been implicated, primary hyperhydrosis appears to be a worldwide problem [3, 13]. **[Q1]**

The natural history of primary palmar hyperhydrosis is poorly documented; however, the onset appears to be during childhood or early adolescence. Most patients present for treatment in the second or early third decades of life, with complaints of psychological, social and occupational disabilities [10, 13]. **[Q2]**

Thoracoscopic sympathectomy has been used effectively to treat several neurological and vascular conditions. The majority of procedures have been performed to treat primary palmar hyperhydrosis [10, 11, 14–33], Raynaud's syndrome [18, 26], Buerger's disease [34], reflex sympathetic dystrophy (causalgia) [18, 26, 34, 35], refractory cardiac arrhythmia [18, 35], and intractable visceral pain [36, 37]. Noninvasive treatments may be used in those patients who have

minor symptoms or who are reluctant to undergo surgery. Topical injection of botulinum has been shown to reduce the amount of local perspiration, but long-term evaluation is needed [38, 39]. Hypnosis, psychotherapy and biofeedback have been beneficial in a limited number of cases [40]. Iontophoresis has attained satisfactory results, but has not yet been studied long term [41–43]. Percutaneous computed tomography (CT)-guided phenol sympathicolysis has shown short-term success, but its long-term failure rate is prohibitive [44]. The topical application of astringents or antiperspirants is considered effective, but only temporarily, thus requiring persistent application; this can often lead to irritation [41, 45]. **[Q3]**

Thoracoscopic sympathectomy has generally been considered to be contraindicated in patients with previous thoracic operations or pulmonary infections who may have dense adhesions that impede thorough visualisation of the sympathetic chain [46]. Nonetheless, it has been shown that fibrous adhesions complicated the procedure in only five per cent of cases [47]. Due to the positioning of the arms throughout the procedure, it is necessary to rule out thoracic outlet syndrome (TOS). If a patient is identified with TOS, the arm should be positioned to minimise the compression of the thoracic outlet, and the upper extremity should be monitored for adequate peripheral pulses throughout the duration of the procedure. **[Q4]**

Possible short-term complications include pneumothorax, haemothorax. subcutaneous emphysema, pleural effusion, and segmental atelectasis [48]. Incomplete reinflation of the collapsed apical lobe will result in postoperative complications from atelectasis and pneumothorax [48]. Rare complications, including false aneurysm of the intercostal artery and inferior brachial plexus injury, have been described in case reports [49–51]. **[Q5]**

Long-term complications include compensatory sweating in the trunk and/or lower extremities, intercostal neuralgia, and Horner's syndrome. Compensatory hyperhydrosis is the most common and unpredictable side effect and is reported in 0–74.5% of cases, with the trunk and lower extremities primarily affected [1, 11, 23, 24, 48, 52]. Anecdotal evidence suggests that the sweating subsides over time [17, 48]. The exact cause is unknown, but an increased number of coagulated segments have correlated with the degree of compensatory sweating [17, 23, 24]. Surgeons should limit their resections to a minimum. Gustatory hyperhydrosis, or facial sweating triggered particularly by spicy foods, has also been reported in up to 56 per cent of patients in some series [53, 54]. Phantom sweating, or a feeling of sweating in the hands without actual sweating, has been reported in 26–59% of patients [55–57].

Intercostal neuralgia may stem from thermal damage to the adjacent intercostal nerve or from compression injury while using large, rigid thoracoports [18]; however, no additional complications of neuralgia have been reported with the change to soft 7-mm ports.

Horner's syndrome, which results from damage to the stellate ganglion, is of most concern to the patient. The condition is transient in 0–23.5% of cases [18], with permanent symptoms occurring in 0–6% of cases [10]. To minimise the risk of Horner's syndrome, the surgeon should take great care to visualise the multiple rami of the stellate ganglion along with their course and direction, and incise immediately caudal to it, above the level of the T2 ganglion. Traction of the stellate ganglion during partial resection of the sympathetic chain may result in transient Horner's syndrome [18]. Heat generation from electrocoagulation, misidentification of the second rib, and injury to the stellate ganglion from pulling

on the sympathetic chain are potential causes of permanent Horner's syndrome. Other possible operative complications include injury to the vagus nerve, phrenic nerve, subclavian artery or subclavian vein. **[Q6]**

Attempts at further reducing access trauma have resulted in the development of ultrafine thoracoscopic instruments. The use of 2-mm ports with two ports of entry has been described in performing sympathectomy or sympathicotomy with less scarring and pain and good outcomes [52, 58]. To improve cosmesis, a two-port axillary and a periareolar procedure has been used in patients presenting for thoracoscopic sympathectomy [59]. A one-port approach has sometimes been used through the axilla, where simple cautery destruction of the chain is performed [60].

Failure of thoracoscopic sympathectomy has resulted primarily from inadequate ablation of the sympathetic nerves or regrowth of the nerve tissue; as such, many investigators recommend obtaining intraoperative histological confirmation of the resected portion of sympathetic chain [18, 22, 25]. Success rates for thoracoscopic sympathectomy limited only to the rami communicantes for the treatment of primary palmar hyperhydrosis range from 71 to 100 per cent, with recurrence rates ranging from zero to 1.5 per cent [15, 19, 28, 38]. For treatment of Raynaud's disease, success and recurrence rates have ranged from 93 to 100 per cent and from zero to 13 per cent, respectively. For the treatment of reflex sympathetic dystrophy, initial success rates have ranged from 86 to 100 per cent, with recurrence rates as high as 62.5 per cent [24, 25]. Results for the treatment of intractable visceral pain yield a success rate of 71–100% and a reported recurrence rate of 33 per cent at 3-months' follow-up [37, 38]. **[Q7]**

Cohen et al. [11, 26, 61] performed thoracoscopic sympathectomies in children and adolescents and concluded that the procedure was safe and effective at a 4-year follow-up. In another series reported by Lin [53] (mean age 14.2 years), in which the T2 or T2 and T3 segments of the sympathetic chain were ablated along with the Kuntz fibres, only one technical failure was reported, with a patient satisfaction rate of 93.5 per cent. The rate of compensatory hyperhydrosis was a disappointing 86 per cent, and the recurrence rates at 1-, 2- and 3-years' follow-up were 0.6, 1.1 and 1.7 per cent, respectively. **[Q8]**

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34. Acute Axillary/Subclavian Vein Thrombosis

Jarlis Wesche, Torbjørn Dahl and Hans O. Myhre

A 34-year-old male motor mechanic was admitted with a 3-day history of severe swelling of the right arm. He had been undertaking physical activity, including weightlifting, training for about 1.5 h four times a week. There was no history of trauma. The patient felt discomfort, but no severe pain in the arm. The superficial veins were distended. The colour of the hand and forearm was slightly cyanotic. The pulses in the radial and ulnar arteries were palpable. No bruits could be heard along the brachial, supraclavicular or axillary arteries. The rest of the examination was unremarkable. The patient did not use any medication.

Question 1

What further diagnostic investigations would you recommend in this patient?

A. Plethysmography.

B. Phlebography.

C. Duplex scanning.

D. Magnetic resonance phlebography.

E. Computed tomography (CT) scanning.

F. X-ray of the chest and thoracic outlet.

G. Venous pressure measurements.

Phlebography revealed a thrombosis of the axillary/subclavian veins (Fig. 34.1). The brachiocephalic vein was patent. There were no signs of skeletal deformities.



Fig. 34.1. Digital subtraction angiography (DSA) phlebogram showing occlusion of the right subclavian vein, but contrast passage to the superior caval vein via jugular/supraclavicular collateral veins (note its relation to the thoracic outlet).

Question 2

Which of the following conditions could lead to axillary/subclavian vein thrombosis?

A. Venous-access catheters.

B. Callus from fractured clavicle or rib.

C. Local tumour/malignancy.

D. Radiotherapy.

E. Trauma to the vein caused by repeated strenuous exercise.

Question 3

Which therapy would you recommend in the acute (2–3 days) phase?

A. Resection of the first rib.

B. Balloon angioplasty of the subclavian vein.

C. Stenting of the subclavian vein.

D. Thrombolysis.

E. Systemic heparin.

F. Thrombectomy.

Question 4

Following thrombolytic therapy for axillary/subclavian vein thrombosis, what percentage of complete lysis can you expect provided the patient is treated within 3 days after start of symptoms?

A. 10%.

B. 25%.

C. 40%.

D. 60%.

E. 80%.

Question 5

A control phlebography revealed a stenosis of the axillary/subclavian vein at the thoracic outlet. There was no residual thrombotic material. At 3 months' follow-up the patient still had pain and discomfort in the arm when going back to his job as a motor mechanic. Which of the following treatment alternatives would you recommend at this stage?

A. Repeated attempt of thrombolytic therapy.

B. Balloon angioplasty and stenting of the subclavian artery.

C. Continued oral anticoagulation therapy.

D. Relief of the thoracic outlet by resection of the first rib.

E. Direct reconstruction of the vein.

Commentary

In patients with acute axillary/subclavian vein thrombosis, it is important to separate primary from secondary thrombosis. Primary thrombosis is also known as Paget–Schrötter syndrome, which is induced by strenuous activity of the arm or venous compression at the thoracic outlet predisposing to thrombosis formation [1–4]. The term "effort thrombosis" is also used for this condition. Men are affected more often than women, and the incidence is higher in the veins of the dominant arm. Secondary axillary/subclavian vein thrombosis could be caused by venous-access catheters, pacemaker wires, malignancies, radiotherapy or compression from local tumour formation. Secondary thrombosis is also seen as a complication of thrombophilia and in patients with dialysis fistulas [5]. **[Q2: A, B, C, D, E]** The preferred therapy may be different in the two groups, and in general a more conserva-

tive attitude is often justified in patients with secondary thrombosis. These patients often have a limited life expectancy due to serious co-morbidities, such as cardiac disease or malignancy, which would also represent a contraindication to thrombolytic therapy. In addition, there is often less need for extensive activity of the upper extremities in this group of patients.

Complications following axillary/subclavian vein thrombosis are swelling, pain and discomfort in the arm prohibiting work or daily-life activities. Furthermore, it has been reported that up to 10 per cent of the patients with axillary/subclavian vein thrombosis develop pulmonary emboli and that it is more common than usually appreciated [5, 6]. Phlegmasia cerulea dolens of the arm is extremely rare and is usually associated with hypercoagulability or malignancies.

In patients with primary axillary/subclavian vein thrombosis, as in our patient, duplex scanning can be performed as a supplement to the clinical examination [6]. However, duplex scanning is operator dependent. If the examination is negative, then phlebography has to be performed anyway. Thus, phlebography, preferably by contrast injection via the basilic vein, should be the gold standard in these cases. Recently, three-dimensional gadolinium-enhanced magnetic resonance phlebography has proven excellent for imaging central veins, and series presenting up to 100 per cent sensitivity and specificity in the accuracy of diagnosing abnormalities in the large central veins have been published [7]. Magnetic resonance phlebography may thus be the future gold standard. A chest X-ray including the thoracic outlet to investigate the possibility of bony deformations is also indicated. **[Q1: B, C, D, F]**

The patient should be evaluated thoroughly for thrombophilia. Blood tests should include a blood count, tests for decreased levels of antithrombin (III), protein C and protein S deficiencies, activated protein C (APC) resistance, antiphospholipid antibodies (lupus anticoagulans) and anticardiolipin antibodies.

As soon as the diagnosis has been established, systemic heparinisation is administered [8]. This should be followed by local thrombolysis using urokinase or preferably recombinant tissue plasminogen activator (rt-PA) unless there are contraindications [9–14]. At introduction of the guidewire, the resistance will indicate the age of the thrombosis and the possibility of obtaining lysis of the thrombotic occlusion. The catheter for application of the thrombolytic agent should be placed within the thrombosis. Usually, a dose of 5 mg rt-PA is given as a bolus, followed by infusion of 0.01 mg/kg body weight/hour for 24–72 h. Injection of thrombolytic material distal to the thrombotic occlusion is less effective since it will usually pass through the collateral veins. **[Q3: D, E]**

Although the most favourable results are obtained in patients with less than 1 week's duration of symptoms [10], an attempt at thrombolysis could be justified even if the symptoms have lasted for 1 month. **[Q4: E]**

After thrombolysis, a repeat phlebography is performed to evaluate whether any intrinsic or extrinsic obstructions of the blood flow are present. Functional phlebography with the arm abducted to 90° with external rotation allows better visualisation of compression of the vein as well as the collaterals. Often a defect is located close to the costoclavicular ligament. Together with hypertrophic anterior scalene and subclavius muscles, this ligament could cause external compression of the vein. The head of the clavicle could contribute to the deformation of the vein. Intrinsic venous stenosis is thought to be due to repetitive trauma damaging venous valves or the endothelium, or producing thickening of the vein wall or intraluminal synechiae, predisposing to thrombosis.

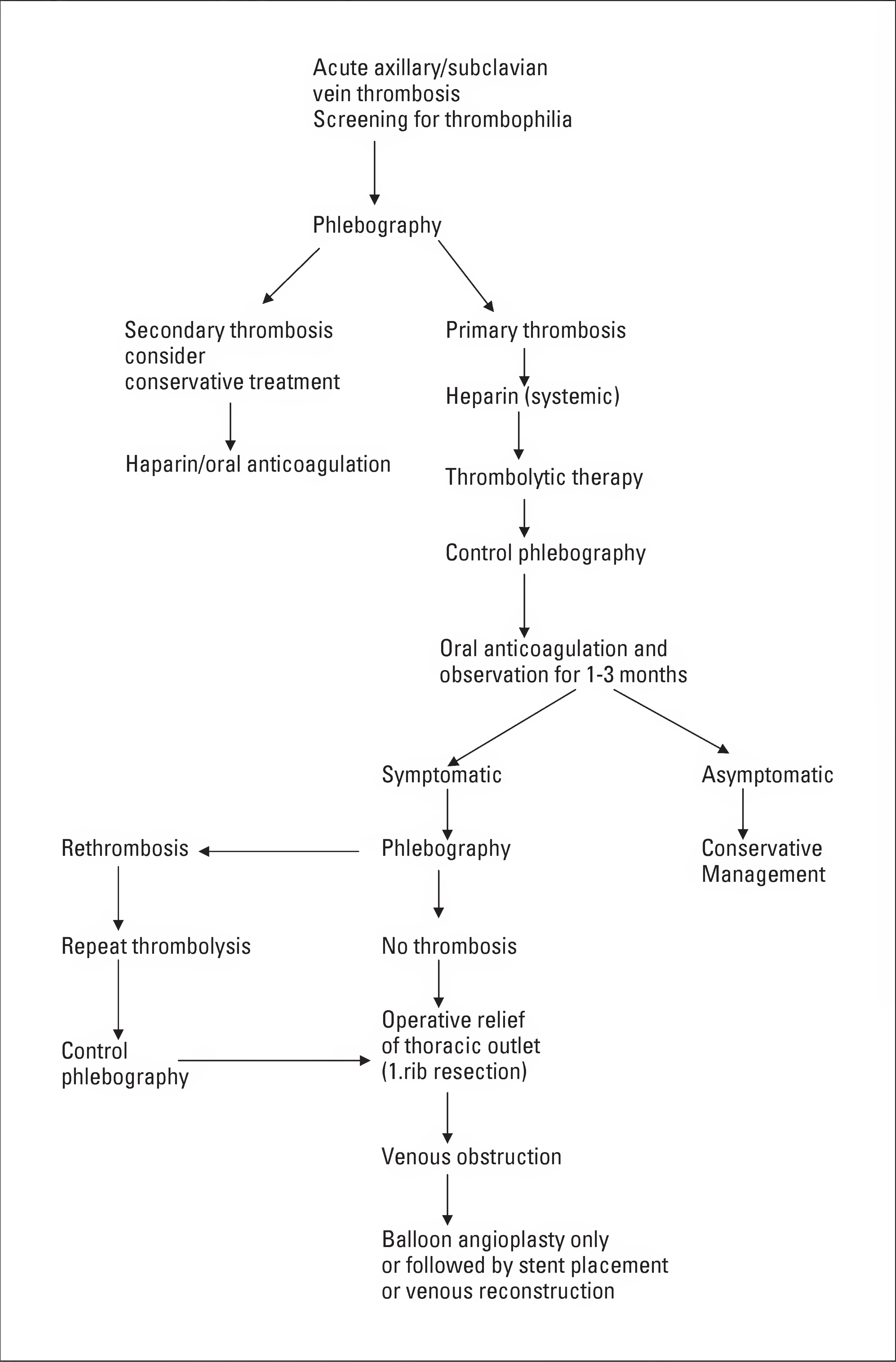


Fig. 34.2. Algorithmic approach to treatment options for acute axillary/subclavian vein thrombosis. Updated 🕮

After thrombolysis, the patient should be on oral anticoagulation for 3–6 months. Heparin is discontinued as soon as the INR (international normalised ratio) has reached therapeutic levels. Following a 3-month follow-up period, the clinical status of the patient should be re-evaluated. Some centres proceed with more radical surgery soon after thrombolysis [11, 15–16]. If the patient is asymptomatic at follow-up, then we do not recommend further treatment. However, this policy is controversial and some centres proceed with decompression of the thoracic outlet routinely.

If the patient is symptomatic and there is a residual stenosis of the subclavian vein caused by either internal or external pathological structures, then the stenosis should not be treated by balloon angioplasty or stenting primarily [11–13, 15, 17]. Whenever these treatment modalities are applied before relief of the thoracic outlet, recurrence of the symptoms will inevitably occur. Furthermore, fracture of the stents has been described because of the "scissors effect" caused by the narrow thoracic outlet [18]. Decompression of the thoracic outlet is obtained by resection of the first rib, including the distal part of the anterior scalenic muscle and fibrous structures adhering to the first rib. **[Q5: D]**

The surgical approach for relieving the thoracic outlet is also controversial: some prefer the transaxillary approach, whereas others use the supra- or infraclavicular approach [3, 5, 11, 15, 16, 19, 20].

After thoracic outlet surgery, a venous obstruction can be treated with balloon angioplasty. This technique is also controversial since there are no randomised studies. Balloon angioplasty could be supplemented with stenting, but the experience with this treatment modality is rather limited. Finally, some authors recommend direct reconstruction by endovenectomy and patch angioplasty for relief of intravenous obstructions in selected cases [3].

In summary, the most effective sequence to restore venous patency and reduce rethrombosis seems to include local thrombolytic therapy, 3–6 months of oral anticoagulation, and then first-rib resection in patients who have significant symptoms at this stage. Occasionally percutaneous transluminal angioplasty (PTA), stent placement or venous reconstruction may be indicated. Following such a staged multidisciplinary treatment (Fig. 34.2), the disability rate after acute axillary/subclavian thrombosis has declined from around 60 per cent to 12 per cent [19].

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35. Raynaud's Phenomenon

Ariane L. Herrick

A 38-year-old female patient presented to the rheumatology clinic with a 3-week history of a painful fingertip ulcer. The pain was so severe that it was keeping her awake at night. For 20 years (since her teens) her hands had been turning white then purple in the cold weather, going red (with tingling) when rewarming. Her feet also felt cold. Her family doctor had told her that this was Raynaud's phenomenon, which was very common. However, each winter her symptoms seemed to be worsening, and even a slight temperature change would bring on an attack. The previous winter she had had some finger ulcers which had, however, been less painful than the current one and which had healed spontaneously. Also of concern to her was that for 6 months the skin of her fingers had felt tight, and she had recently been experiencing some difficulty swallowing, with heartburn. There was no past medical history of note. She had smoked five cigarettes a day for 2 years. There was no history of chemical exposure nor of use of vibratory equipment.

Question 1

Which symptoms suggest that this is *not* primary (idiopathic) Raynaud's phenomenon?

A. Onset of Raynaud's phenomenon age 18 years.

B. The feet were affected as well as the hands.

C. Development of digital ulcers.

D. The skin of the fingers felt tight.

E. She was a smoker.

On examination she had a healing ulcer at the tip of the left middle finger (Fig. 35.1). The fingertip was extremely tender. She had mild skin thickening of the



Fig. 35.1. Fingertip ulcer in a patient with systemic sclerosis.

fingers (sclerodactyly) but elsewhere the skin was normal. She had digital pitting of the right index and middle fingers. There were no other abnormal findings.

Question 2

What investigations would you perform?

A. Full blood count and erythrocyte sedimentation rate.

B. Angiography.

C. Testing for antinuclear antibody (ANA).

D. Testing for anticentromere antibody.

E. Nailfold capillaroscopy.

Full blood count and erythrocyte sedimentation rate (ESR) were normal. On immunological testing she was strongly antinuclear antibody (ANA) positive (titre 1/1000) and she was anticentromere antibody positive. Chest X-ray showed no cervical rib. Hand X-rays were normal. Nailfold microscopy was abnormal, showing widened, dilated loops with areas of avascularity (Fig. 35.2).

Question 3

What is the diagnosis?

A. Limited cutaneous systemic sclerosis ("CREST" syndrome).

B. Hyperviscosity state, for example secondary to malignancy.

C. Extrinsic vascular compression.

D. Atherosclerosis.

E. Buerger's disease.



Fig. 35.2. Typical appearances on nailfold microscopy in systemic sclerosis – several capillary loops are dilated, with areas of avascularity.

Question 4

Which of the following are true of systemic sclerosis (also termed "scleroderma"):

A. Digital pitting is a characteristic feature.

B. Males are more commonly affected than females.

C. The two subtypes – limited cutaneous and diffuse cutaneous – are separated on the basis of the extent of the skin involvement.

D. Raynaud's phenomenon often precedes the diagnosis of limited cutaneous systemic sclerosis by many years.

E. Anticentromere antibody is a risk factor for severe digital ischaemia requiring amputation.

The diagnosis of limited cutaneous systemic sclerosis was explained to the patient. She was told that her Raynaud's phenomenon and her upper gastrointestinal symptoms were most likely related, and that some checks of her cardiorespiratory function would be arranged on a routine basis.

Question 5

How would you have treated her Raynaud's phenomenon had you seen her 6 months previously, when there was no digital ulceration?

A. Avoidance of cold exposure.

B. Low dose prednisolone.

C. Stop smoking.

D. Nifedipine (sustained release).

E. Biofeedback.

The patient was prescribed nifedipine (sustained release) and a course of flucloxacillin. When reviewed one week later, the fingertip had deteriorated and some of the tissue had become necrotic, with surrounding erythema.

Question 6

What would you do now?

A. Admit to hospital for intravenous prostanoid therapy.

B. Intravenous antibiotics.

C. Debridement of the ulcer.

D. Cervical sympathectomy.

E. Anticoagulation.

The patient was admitted for intravenous antibiotic therapy, intravenous prostanoid infusions, and a vascular opinion. The fingertip was debrided. The patient was discharged home 6 days later, with instructions to dress warmly, avoid cold exposure, and to seek medical advice early should any further ulcers develop.

Commentary

Raynaud's phenomenon – episodic digital ischaemia usually in response to cold exposure or stress – can be either *primary* (idiopathic) or *secondary* to a number of different diseases/conditions, including connective tissue disease (most characteristically systemic sclerosis), external vascular compression (as with a cervical rib), vibration exposure, hyperviscosity, drug treatment (for example beta-blockers, ergotamine) and occupational chemical exposure. The terminology is confusing: primary Raynaud's phenomenon was previously termed "Raynaud's disease", and secondary Raynaud's phenomenon "Raynaud's syndrome". However, "primary Raynaud's phenomenon" and "secondary Raynaud's phenomenon" are now the preferred terms [1].

The pathophysiology of Raynaud's phenomenon (either primary or secondary) is poorly understood. Raynaud's phenomenon can occur because of abnormalities in vascular structure, vascular function, or the blood itself [2]. These are interdependent and may occur together, as in systemic sclerosis when structural vascular problems inevitably impair vascular function, and platelet and white blood cell activation, together with impaired fibrinolysis, are also thought to contribute to pathophysiology. It is generally accepted that primary Raynaud's phenomenon is mainly vasospastic and does not progress to irreversible tissue damage. In contrast, Raynaud's phenomenon secondary to connective tissue disease such as systemic sclerosis is associated with structural vascular abnormality, and patients often develop ulceration, scarring, and even gangrene necessitating amputation.

The vascular surgeon is likely to encounter patients with Raynaud's phenomenon for two main reasons:

1. Diagnosis. Why does this patient have episodic digital ischaemia?
2. Treatment of a critically ischaemic digit, or of severe Raynaud's phenomenon unresponsive to medical therapy.

The onset of primary Raynaud's phenomenon is most commonly in the teens or twenties: onset in later years should always raise the suspicion of an underlying cause. Women are more commonly affected. For Raynaud's phenomenon to be primary, there should be no clinical features of underlying connective tissue disease or other disease/disorder (including absence of digital pitting or sclerodactyly), there should be no digital ulceration or gangrene, the ESR should be normal, testing for ANA negative (titre <1/100) and the nailfold capillaries should be normal [1]. **[Q1: C, D]** In the absence of any worrying features in the history and examination, the usual investigation screen therefore comprises a full blood count and ESR, testing for ANA, nailfold capillaroscopy and, if there is any question of a cervical rib, a chest or thoracic outlet X-ray. Anaemia and/or a high ESR may indicate an underlying connective tissue disease or other illness. However, a normal haemoglobin level and ESR (as in our patient) do not exclude a diagnosis of systemic sclerosis, in which the vascular abnormalities are primarily non-inflammatory [3]. In primary Raynaud's phenomenon, the nailfold capillaries should be fairly regular "hair-pin" loops as opposed to the abnormal dilated loops, with areas of loop drop-out, that are characteristic of systemic sclerosis [4].

Other investigations are indicated by the history and examination. For example, if there is sclerodactyly (scleroderma of the fingers) and/or digital pitting (Fig. 35.3), which are both characteristic of systemic sclerosis, then anticentromere antibodies and antibodies to topoisomerase (anti-Scl-70 antibodies) should be looked for. These antibodies are highly specific for systemic sclerosis [5]. If there is any

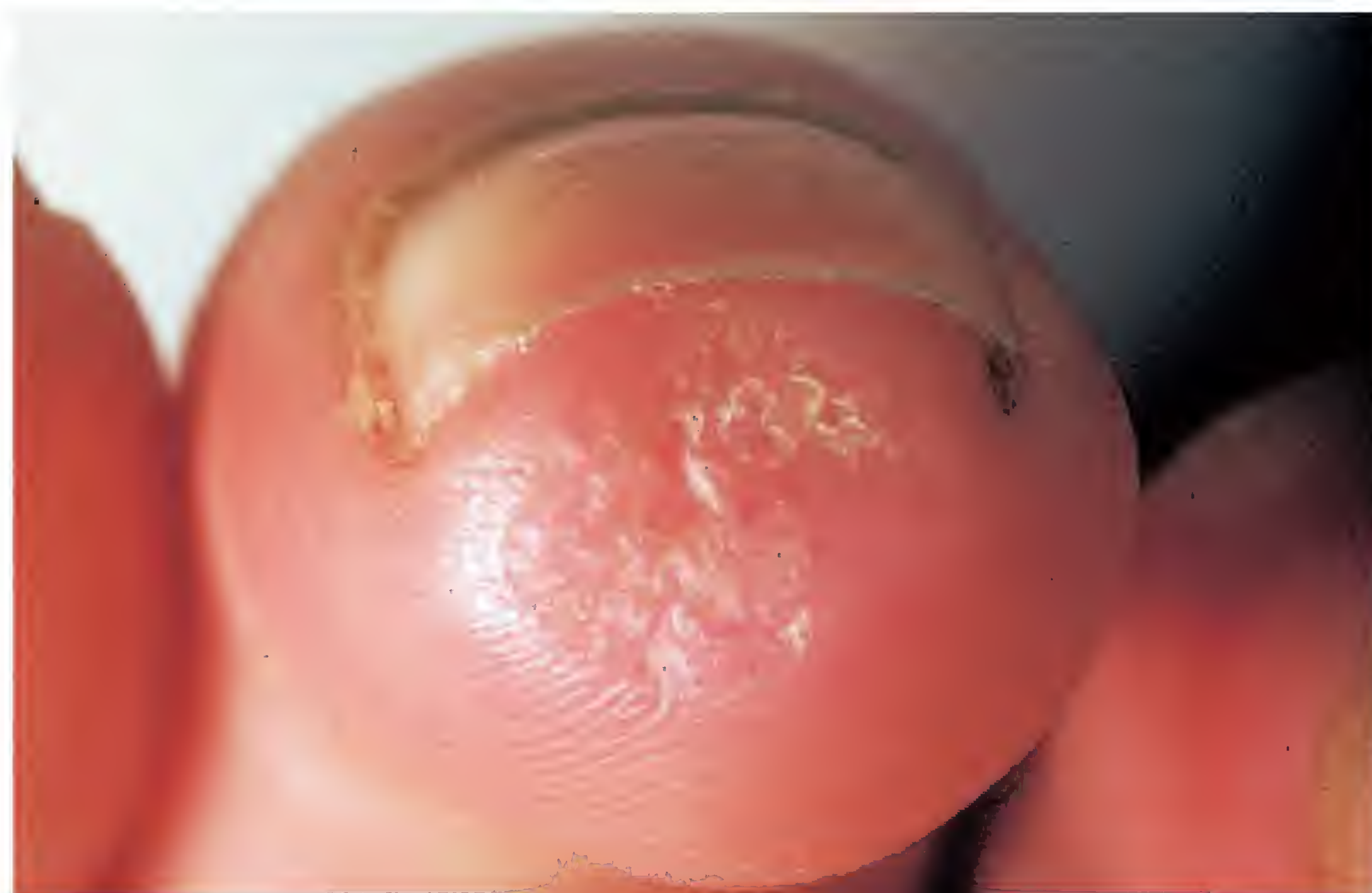


Fig. 35.3. Digital pitting in a patient with systemic sclerosis. 🕮

question of a proximal vascular obstruction (absent peripheral pulses) then angiography should be considered, but in the majority of patients with systemic sclerosis and digital ischaemia this is not necessary. **[Q2: A, C, D, E]**

Systemic sclerosis, similarly to primary Raynaud's phenomenon, is more common in women than in men. There are two main subtypes of systemic sclerosis – limited and diffuse cutaneous – defined on the basis of the extent of the skin involvement. In patients with limited cutaneous disease (previously termed CREST – calcinosis, Raynaud's, oesophageal dysmotility, sclerodactyly, telangiectases), only the skin of the extremities and face is thickened, whereas in those with diffuse cutaneous disease there is proximal skin thickening, involving proximal limbs and/or trunk [6]. The patient described has clinical features typical of limited cutaneous disease: Raynaud's phenomenon preceding the diagnosis of systemic sclerosis by a number of years, sclerodactyly, digital pitting, and upper gastrointestinal problems. **[Q3: A]** Patients with limited cutaneous disease typically have more severe digital vascular disease than patients with diffuse cutaneous disease, and anticentromere antibody is predictive of severe digital ischaemia [7]. **[Q4: A, C, D, E]**

Treatment of Raynaud's phenomenon is initially conservative – keeping warm, avoiding cold exposure, and refraining from smoking (smoking is a risk factor for severity of digital ischaemia in patients with systemic sclerosis [8]). If these measures do not suffice, then a vasodilator is prescribed, usually a calcium channel blocker [9, 10]. There is no role for steroid therapy in most patients with systemic sclerosis (and steroids are relatively contraindicated in patients with diffuse cutaneous disease). Biofeedback has gained considerable attention but was not found to be effective in a randomised trial of primary Raynaud's phenomenon [11]. **[Q5: A, C, D]** If a patient has very severe digital ischaemia, with or without digital ulceration, then the patient should be admitted for intravenous prostanoids [12] and, if there is any question of infection, then intravenous antibiotics are also indicated.

The vascular surgeon is likely to be called to see a patient with severe Raynaud's (often in the context of systemic sclerosis) because of either non-healing ulceration or because of very severe (sometimes critical) ischaemia. The reduced blood supply



Fig. 35.4. Digital pulp calcinosis in a patient with systemic sclerosis – there is a risk that this deposit will ulcerate. 📖

impairs ulcer healing. Debridement often aids healing. However, a proportion of patients come to amputation. Some patients have calcinosis at the site of the ulceration, and so this may be a complicating factor (Fig. 35.4). Severe ischaemia often coexists with ulceration. Cervical sympathectomy is no longer advocated for upper limb Raynaud's phenomenon. Recently digital sympathectomy has attracted interest for the treatment of severe digital ischaemia in patients with systemic sclerosis [10, 13]. Digital sympathectomy is unlikely to be indicated at this stage in our patient, unless things do not settle with intravenous prostanoids, antibiotics and debridement. At present there is no evidence base for anticoagulation in patients with systemic sclerosis and digital ischaemia and/or ulceration although the possibility of an underlying coagulopathy, for example antiphospholipid syndrome, should always considered in patients presenting with digital ischaemia. **[Q6: A, B, C]**

Finally, although the vascular abnormalities in systemic sclerosis are predominantly microvascular, an increased prevalence of large vessel disease in patients with systemic sclerosis has recently been reported [14]. Thus the possibility of a proximal obstruction should always be considered in patients with systemic sclerosis presenting with an ischaemic digit.

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36. Aortofemoral Graft Infection

Christopher P. Gibbons

A 66-year-old man, an ex-smoker with hypertension and hypercholesterolaemia, had undergone a Dacron bifurcated aortic graft and bilateral ureteric stents for an inflammatory aortic aneurysm with ureteric obstruction at another hospital 4 years previously. The left limb of the graft had been anastomosed to the common femoral artery and the right limb to the common iliac bifurcation. Postoperatively he had suffered a mild groin wound infection, which had healed with antibiotics. At follow-up he complained of left calf and thigh claudication. On examination, he appeared generally well with a midline abdominal scar and a left vertical groin scar. He had good right femoral pulse but an absent left femoral pulse.

Question 1

What should be the first investigation?

A. Intra-arterial digital subtraction angiography.

B. Duplex ultrasound scan of the aortic graft.

C. ^{99}Technetium-labelled leucocyte scan.

D. CT angiography of the graft.

E. Erythrocyte sedimentation rate.

A duplex scan showed an occluded left limb of the aortic graft with patent common femoral arteries. There was no evidence of any stenosis of the left common femoral artery but a perigraft fluid collection was noted around the intra-abdominal portion of the graft.

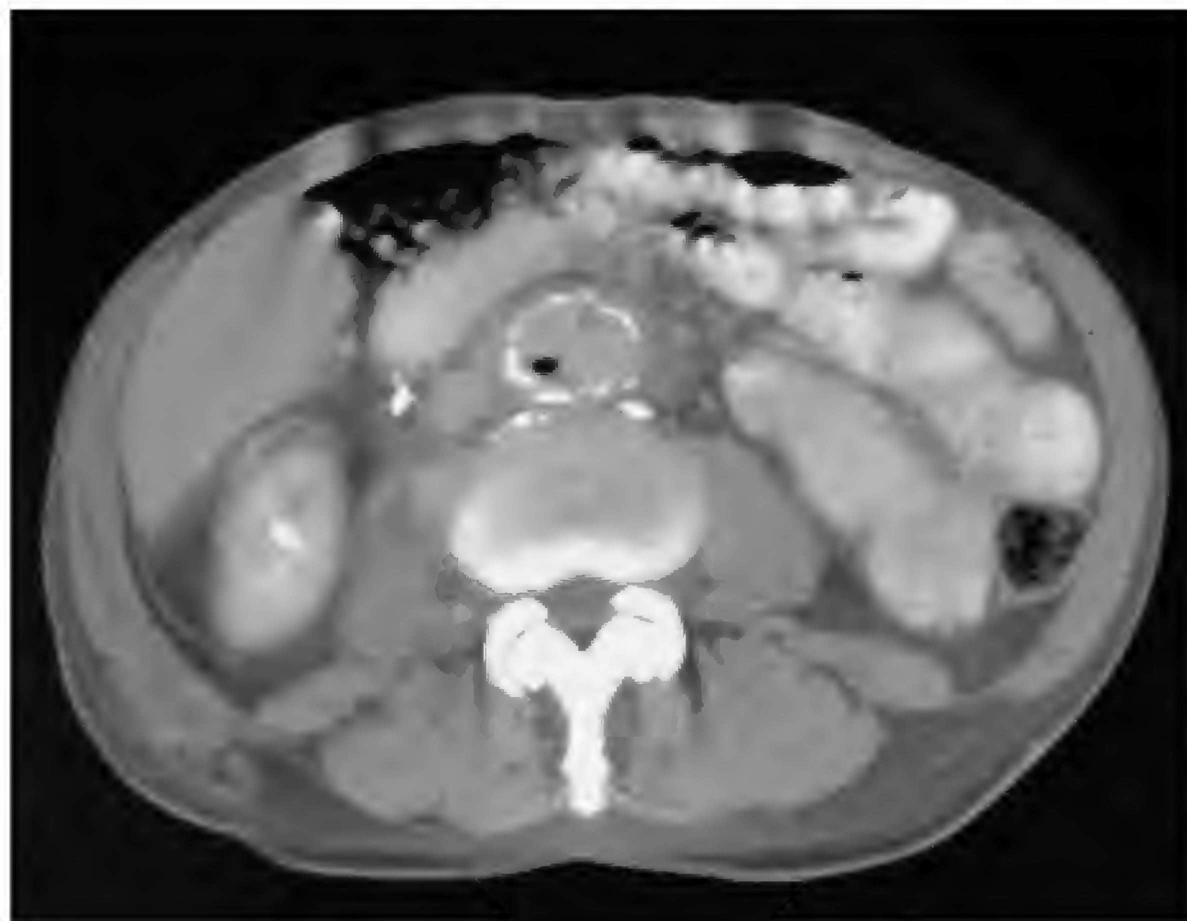


Fig. 36.1. CT scan of aortic graft showing fluid and a gas bubble around the graft.

Question 2

What further investigations should be performed?

A. CT scan of the graft.

B. Digital subtraction angiography.

C. ^{99}Technetium-labelled leucocyte scan.

D. Erythrocyte sedimentation rate.

E. Aspiration of the collection.

A CT scan confirmed the presence of fluid and gas around the intra-abdominal portion of the graft and the occlusion of the left limb, indicating graft infection (Fig. 36.1). Digital subtraction angiography (Fig 36.2) confirmed the occluded left limb of the aortic graft and showed a stenosis at the origin of the right graft limb, presumably as a result of external compression. Aspiration of the perigraft collection would have allowed preoperative bacterial culture but was considered to be too difficult to perform safely.

Question 3

Having confirmed the presence of infection what is the best option for management?

A. Prolonged antibiotic therapy.

B. Drainage of the perigraft pus under anaesthesia.

C. Insertion of gentamicin beads.



Fig. 36.2. Intra-arterial digital subtraction angiography (DSA) of the aortoiliac region.

D. Excision of the graft.

E. Excision of the graft with in situ replacement with an antibiotic bonded graft.

F. Graft excision and extra-anatomical prosthetic bypass.

G. Graft replacement with autologous vein.

H. Graft replacement with an aortic allograft.

In situ replacement with autologous vein was chosen because of the reduced risk of persistent infection.

Question 4

Which autologous veins may be used for aortoiliac or aortofemoral graft replacement?

A. Long saphenous vein.

B. Cephalic vein.

C. Femoropopliteal vein.

D. Iliac vein.

Femoropopliteal vein was used as it is ideally suited to supra-inguinal graft replacement as it is relatively thick-walled, is of adequate diameter and has sufficient length.

Question 5

What further preoperative investigations should be performed?

A. Plain abdominal X-ray.

B. Bone scan.

C. MRI scan of the abdomen.

D. Duplex scan of the femoral veins.

E. Repeat abdominal ultrasound scan.

A duplex scan of the femoral veins confirmed that they were patent and of adequate calibre. The patient was operated on electively on the next available operating list.

Question 6

What other preoperative preparations should be undertaken?

A. Routine full blood count.

B. Urea and electrolyte estimation.

C. Chest X-ray and electrocardiogram (ECG).

D. Compression stockings.

E. Subcutaneous heparin.

F. Combination antibiotic therapy.

Routine blood investigations, chest X-ray and ECG were all performed, and in view of the magnitude of the procedure, echocardiogram and lung function tests were also ordered. They were all satisfactory. Because the bacteriology of the infection was not known preoperatively in this patient, intravenous combination antibacterial therapy with teicoplanin, ciprofloxacin, co-amoxiclav and metronidazole was given immediately before surgery.

Question 7

How should the operation be performed?

A. Laparotomy, excision of the aortic graft, harvesting of the femoral veins and graft replacement.

B. Harvesting of femoral veins followed by laparotomy, excision of the infected graft and replacement with femoral vein.

C. Laparotomy and exposure of the infected graft, then femoral vein harvest followed by graft replacement.

The anaesthetised patient was catheterised, prepared and draped so that the abdomen and both legs were exposed. First, both superficial femoral veins were simultaneously dissected out by two operative teams and the branches divided between clips from the profunda femoris vein to the knee joint. The femoral veins were left in situ whilst the abdomen was opened, exposing the graft and obtaining control of the proximal infrarenal aorta and the right common iliac bifurcation. The graft was encased in fibrous tissue, making dissection difficult and hazardous. The underlying prosthesis showed poor tissue incorporation and there was a localised abscess between the graft and the duodenum, which was evacuated and cultured. The left groin was exposed, obtaining control of the common femoral artery, its branches and the profunda femoris artery. After systemic heparinisation, the vessels were clamped and the infected graft excised and sent for culture. The graft bed was washed repeatedly with povidone iodine and hydrogen peroxide. One femoral vein was excised, reversed and inserted end-to-end from the infrarenal aorta to the right common iliac artery bifurcation using 4/0 polypropylene sutures. Size discrepancy at the aortic anastomosis was overcome by "fishmouthing" the end of the vein to prevent the angulation associated with spatulation (Fig. 36.3). The other femoral vein was reversed and anastomosed end to side to the intra-abdominal part of the vein graft and to the left common femoral artery (Fig. 36.4). Both veins were led

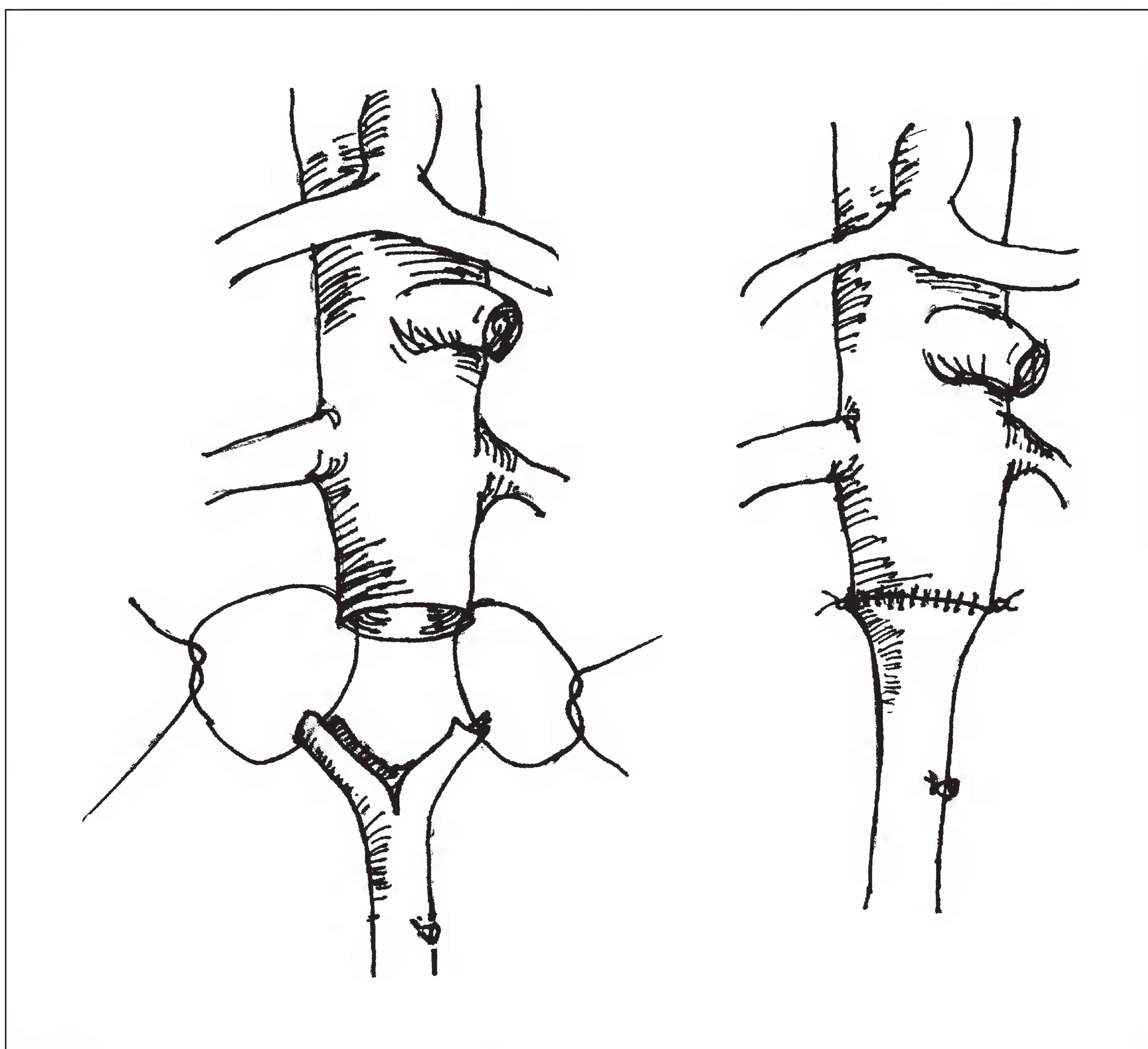


Fig. 36.3. "Fishmouthing" the femoral vein to equalise diameter with the aorta.

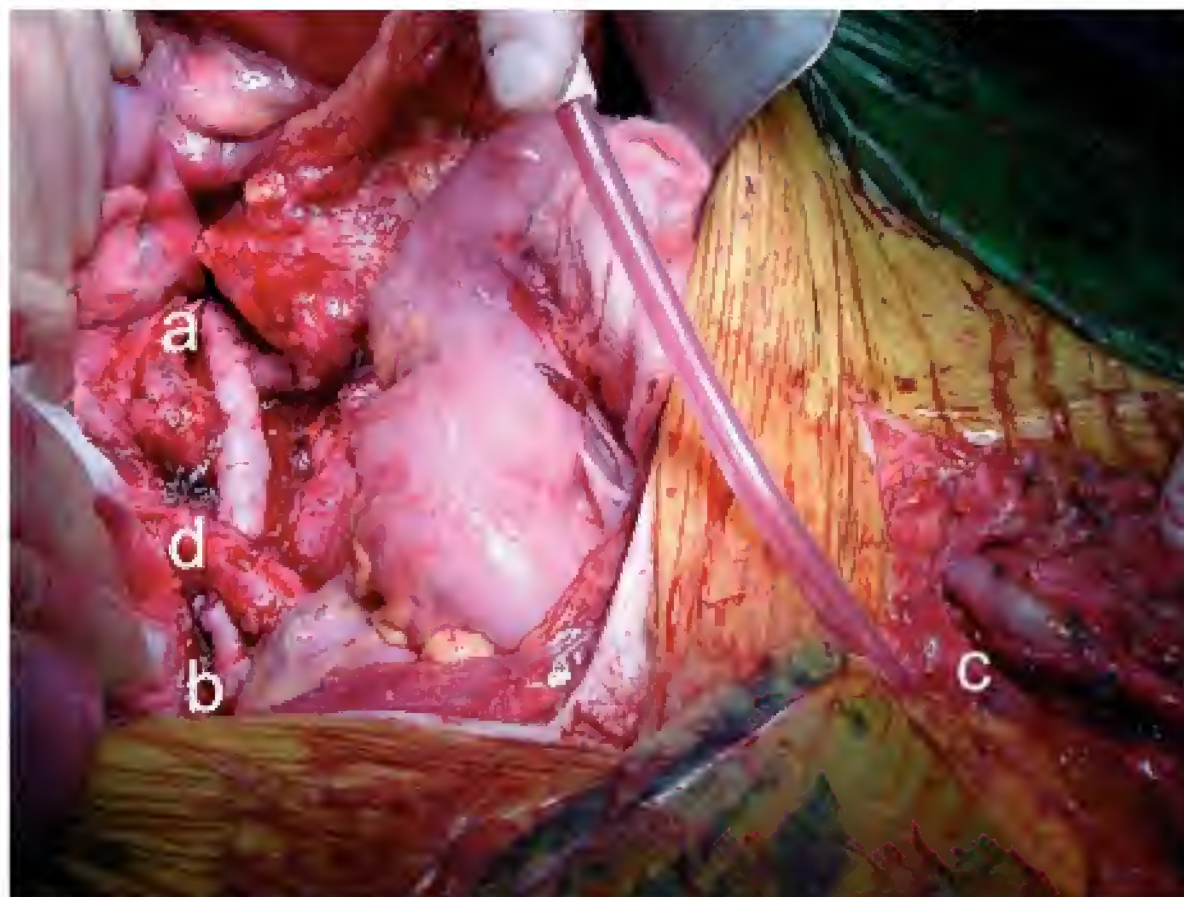


Fig. 36.4. Femoral vein reconstruction from the infrarenal aorta **a** to the right iliac **b** and left common femoral artery **c**. The right ureter **d** overlies the right limb of the graft.

through a fresh tunnel and surrounded by greater omentum to avoid contact with the bed of the infected graft. The arterial anastomoses were covered by gentamicin-impregnated collagen foam and the wounds were closed with suction drainage. Antibiotic prophylaxis and low molecular weight heparin were continued postoperatively. Despite the copious pus around the graft, no organisms were grown in the laboratory. Combination antibiotic therapy was stopped after 7 days but co-amoxiclav was continued empirically for a further 5 weeks.

Question 8

If the patient had presented with an exposed prosthetic graft in the groin how would this have altered management?

A. Prolonged antibiotic therapy.

B. Use of vacuum dressings.

C. Simple coverage with a muscle flap without graft replacement.

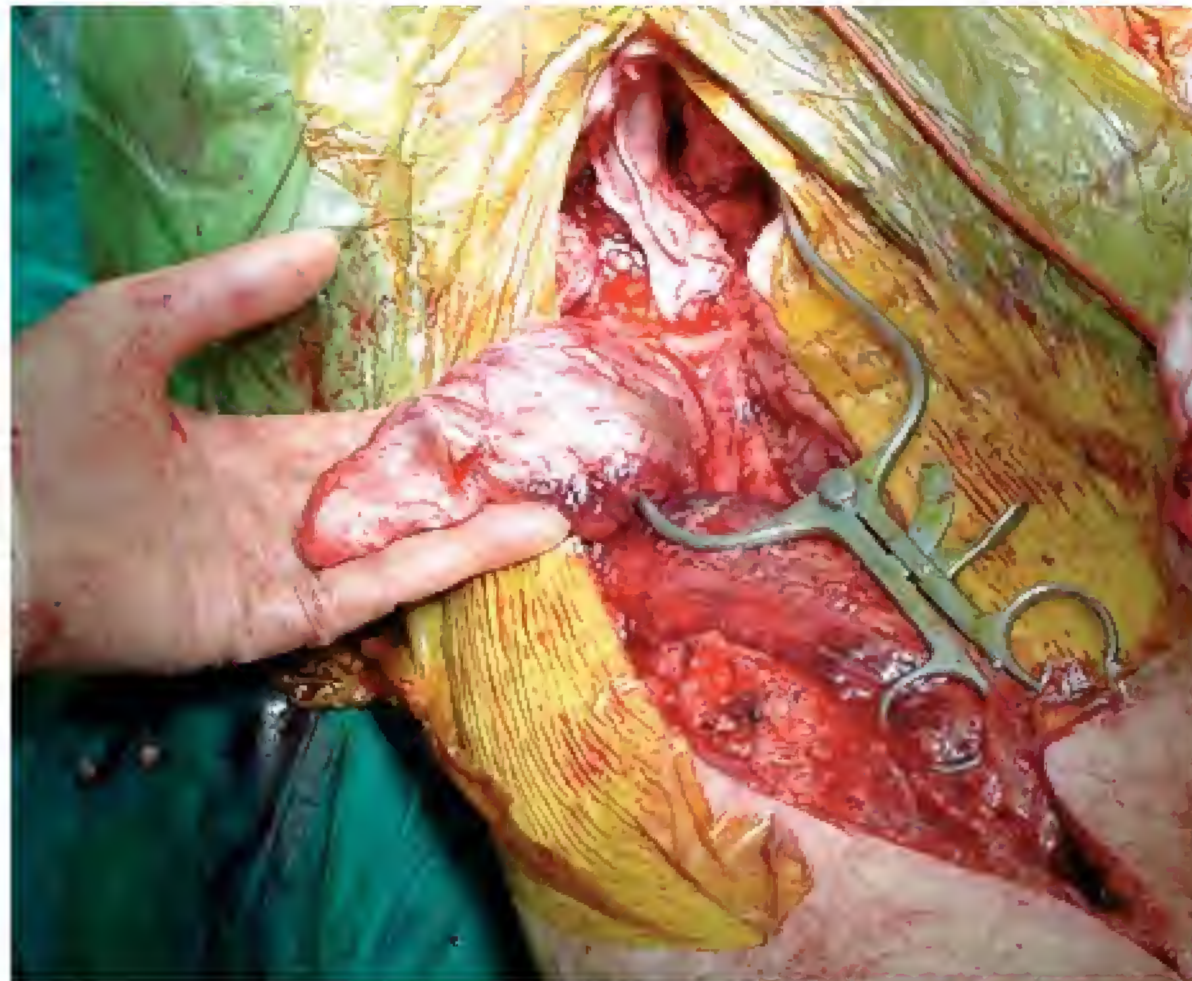
D. Addition of a muscle flap to graft replacement with autologous vein.

Exposed grafts present a difficult problem for achieving skin closure and the addition of a local muscle flap in the groin to graft replacement with autologous (femoropopliteal) vein is the most certain method of cure. The author's preference is a rectus femoris flap for this (Fig. 36.5).

Question 9

What complications might occur following this operation?

A. Anastomotic haemorrhage.



a



b

Fig. 36.5. Rectus femoris muscle flap to cover a femoral anastomosis. **a** After mobilisation of the rectus femoris muscle (the femoral anastomosis is obscured by a sheet of gentamicin-impregnated collagen foam). **b** The muscle now overlies the anastomosis.

B. Graft rupture.

C. Femoral deep vein thrombosis (DVT).

D. Limb swelling.

E. Seroma in the thigh.

F. Intestinal obstruction.

G. Wound infection.

The patient had the most common complication of this operation, which was a large seroma in the left thigh and a smaller one in the right thigh despite prolonged suction drainage. These were aspirated repeatedly and resolved after 3 weeks, although in other cases reoperation and reinsertion of a drain may be required. Intestinal obstruction is no more common following this operation than other abdominal procedures.

The patient otherwise made a good recovery and was discharged from hospital at 14 days. The graft and pus swabs were sterile so most of the antibiotics were stopped at 1 week but the co-amoxiclav was continued for 6 weeks on empirical grounds.

Question 10

What routine follow-up investigations should be performed?

A. Abdominal CT scan.

B. Abdominal ultrasound.

C. Graft duplex scans.

D. Abdominal X-ray.

E. Erythrocyte sedimentation rate (ESR).

Routine 3-monthly duplex scans were performed over 1 year for vein graft surveillance. The patient remains well without further intervention at 5 years.

Commentary

Aortic graft infection is thankfully rare, occurring in 1–5 per cent of reconstructions [1], but is one of the most feared complications in vascular surgery because of its high mortality and morbidity [2]. In a UK multicentre audit of 55 graft infections 31 per cent died, 33 per cent underwent amputation and only 45 per cent left hospital alive without amputation [3]. If left untreated there is a high risk of graft occlusion and anastomotic haemorrhage, which may lead to aorto-enteric fistula. Prompt curative treatment is therefore wise in patients sufficiently fit to withstand major surgery.

Graft infection may present at any time from a few days to many years after surgery. It can follow a wound infection, particularly in the groin where wound breakdown may result in exposure of the graft, or it may present later with a perigraft fluid collection or sinus at the femoral anastomosis. Infection of wholly intra-abdominal grafts may present with backache and fever but more often remain undetected until anastomotic haemorrhage or graft thrombosis occurs. Duplex ultrasound scanning or CT angiography is wise in all cases of graft thrombosis not only to confirm the occlusion but also to demonstrate any perigraft fluid which would indicate graft infection. **[Q1: B, D]**

The most common causative organism is *Staphylococcus aureus* in most series and such infections tend to present in the early postoperative period. Methicillin-

resistant strains (MRSA) are said to be particularly virulent and have been associated with a high mortality in some series [3–5]. *Staphylococcus epidermidis* infections tend to be less virulent and often present many years later. They produce a slime or biofilm around the graft or occasionally thin pus. Isolation of *Staph. epidermidis* is more difficult and may require agitation of the extirpated graft with ultrasound to release it for culture. Other infections are caused by coliforms, *Salmonella*, *Serratia*, *Pseudomonas*, enterococci, streptococci or *Bacteroides* [6]. Gram-negative organisms may be more likely to present with anastomotic haemorrhage [7]. In many cases no causative organism can be isolated despite obvious infection. Possible causes of this are previous antibiotic administration or failure to isolate *Staphylococcus epidermidis*.

A preoperative diagnosis of graft infection is usually secured by ultrasound followed by CT or MRI. Aspiration of the perigraft fluid may secure a bacteriological diagnosis prior to surgery, although in many cases the responsible organism cannot be isolated. Fluid is often present in the aneurysmal sac after aortic aneurysm replacement and can be seen in smaller quantities around an aorto-bifemoral prosthesis performed for occlusive disease on ultrasound or CT for a few weeks after surgery. However, persistence of fluid around an aortic prosthesis for more than 3–6 months after surgery is highly suggestive of infection. Similarly, perigraft gas may be present for up to 10 days after surgery but indicates infection beyond this time [9, 10].

If a groin abscess develops in relation to an aorto-bifemoral graft, aspiration under aseptic conditions in the clinic will confirm the presence of graft infection and may provide preoperative bacteriology. Perigraft fluid or gas may be absent in low-grade chronic infection or if a sinus in one or other groin allows the pus to escape. Exploration of a sinus under anaesthesia will demonstrate a connection with the infected graft and gently passing a bougie alongside the graft will determine whether or not the infection is confined to the anastomosis. If there is no sinus or perigraft fluid, a ^{99}technetium-labelled leucocyte scan may demonstrate increased activity over an infected graft [11]. However, this investigation has poor sensitivity and specificity and is only useful for chronic graft infection as increased leucocyte adherence is demonstrated by most prostheses for up to 6 months after insertion. The ultimate diagnosis of graft infection is made at operation by the lack of tissue incorporation into knitted Dacron or polytetrafluoroethylene (PTFE) prostheses and the presence of perigraft pus from which organisms may be cultured. Preoperative angiography is helpful for operative planning by delineating the vascular anatomy but adds no useful information about the presence of graft infection. **[Q2: A, B]**

There are multiple treatment options: Antibiotic therapy may buy time, but is rarely curative because the graft acts as a foreign body rendering the responsible organisms inaccessible to antibiotics. There have been occasional reports of successful treatment by drainage of the abscess around the graft followed by irrigation with antibiotic or iodine solutions [12–14] or implantation of gentamicin-impregnated beads or foam but these are anecdotal [15, 16]. Simple excision of an aortic graft is unwise unless it has already occluded without critical ischaemia as subsequent limb loss or severe lower body ischaemia is likely. Excision of the infected graft with debridement and replacement with a rifampicin-bonded or silver-impregnated graft has been advocated [17–20] but most would reserve this for chronic low-grade infections because of the risk of reinfection of the new graft. Despite their in vitro effectiveness [21] encouraging individual series, there is no

convincing clinical evidence that either rifampicin-bonding or silver-impregnated Dacron grafts are less susceptible to reinfection after replacement of infected grafts. Moreover, randomised clinical studies have failed to show that either rifampicin-bonding or silver impregnation prevents primary infection in vascular grafts, although these studies were somewhat underpowered [22, 23].

An alternative approach, which avoids direct reimplantation of prosthetic material, is in situ replacement with fresh or cryopreserved aortic allografts. The reported results have been variable but in all series there have been instances of early or late graft disruption or aneurysm formation particularly with fresh allografts and when used for aorto-enteric fistula [24–29].

Until the last 5 years, the mainstay of treatment has been excision of the graft with extra-anatomical reconstruction. For infected aortoiliac grafts reconstruction can be performed with an axillo-bifemoral or bilateral axillofemoral grafts. However, in those patients with infected aorto-bifemoral grafts, the lower anastomosis must be performed at the level of the superficial femoral or popliteal artery to avoid placing the new graft in an infected field. Good results can be obtained with this approach but there remains a 10–15 per cent risk of graft reinfection [2, 30–32]. If this option is used, the extra-anatomic bypass should be performed before graft excision to reduce the risk of irreversible limb ischaemia and amputation [2].

More recently, Claggett [33, 34] and Nevelsteen [35, 36] independently advocated aortic replacement with femoropopliteal veins for infected grafts. Femoropoliteal veins are much wider and thicker-walled than long saphenous or arm veins and have adequate length, making them ideal for aortoiliac reconstruction. Iliac veins are too short and their excision would result in severe limb swelling. Results were excellent with reduced mortality, limb loss and reinfection rates. Subsequently other authors have confirmed the effectiveness of this approach in eliminating reinfection, with mortality and amputation rates similar to or lower than reports using other techniques [37, 38]. This is now recognised as the procedure of choice in most situations. The procedure is demanding and may take several hours to perform but can be made easier by the use of two or more operative teams working together. Femoral veins may be harvested even after the removal of the long saphenous vein but it is generally advised that the profunda femoris vein should be left intact and that the popliteal vein should not be removed below the knee joint [39, 40]. Fears of venous morbidity from femoral vein harvest have not been borne out in practice although Valentine reported an 18 per cent incidence of prophylactic or therapeutic fasciotomy for compartment syndrome [41]. However, neither Nevelsteen [42] nor the present author has found this necessary. Femoral vein harvest should be the initial step in the operation to avoid prolonged abdominal exposure or aortic clamping. Partial graft replacement is best avoided, as the remaining graft usually requires later replacement [43]. **[Q3: E, F, G, H] [Q4: C] [Q7: B]** If femoropopliteal aortic reconstruction is planned, it is wise to perform a preoperative venous duplex scan of the legs to confirm that the femoral veins are patent and of adequate calibre (1 cm). **[Q5: D]**

Whichever technique is used, the importance of adequate debridement, antiseptic washouts and drainage cannot be overstressed. Combination antibiotic cover (beginning immediately preoperatively) to cover any cultured organism and the common pathogens is essential to eliminate infection and prevent catastrophic haemorrhage from anastomotic breakdown. Routine preoperative investigations such as full blood count, urea and electrolyte estimation, chest X-ray and ECG are indicated. Compression stockings are used by some surgeons after femoropopliteal

vein harvest to limit ankle swelling but cannot be used intraoperatively. Subcutaneous heparin is used postoperatively but not preoperatively as systemic heparinisation is used routinely on aortic clamping. **[Q6: A, B, C, F]**

In cases where a prosthetic graft has been exposed in the groin or the tissues overlying a femoral anastomosis are deficient, a local muscle flap is wise to protect the new femoral anastomosis. **[Q8: D]** A sartorius rotation flap may be used provided the graft does not extend in front of the inguinal ligament as in femoral-femoral or axillofemoral grafts [44, 45]. Gracilis and rectus abdominis flaps or omental pedicles have also been described [46–51]. A rectus femoris flap is very quick and easy to prepare and will cover any femoral anastomosis with ease [38]. In occasional localised low-grade graft infections with exposure of the prosthesis in the groin, a simple muscle flap may be successful without excision of the graft but there is a high rate of recurrent infection.

Despite the magnitude of the procedure, graft replacement with femoral vein gives excellent results with reported mortalities in the region of 10 per cent, amputation rates of 10 per cent and no reinfection. Possible complications include anastomotic haemorrhage, iliac venous thrombosis, limb swelling, seroma, intestinal obstruction and wound infection. Limb swelling usually occurs but is rarely excessive and is easily controlled by elevation provided the profunda femoris vein is preserved and the popliteal vein is not harvested below the knee. Since the deep veins have been excised, lower limb DVT is unlikely. Wound and graft infection or anastomotic haemorrhage is similarly infrequent provided adequate antibiotic cover is used. **[Q9: A, D, E, F, G]**

Routine duplex surveillance of the aortofemoral vein grafts is wise as late graft stenosis due to intimal hyperplasia is common [38]. **[Q10: C]**

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37. Aortoenteric Fistulas

David Bergqvist

A 63-year-old woman, who was a smoker, presented with severe intermittent claudication for a couple of years. Her walking distance had decreased gradually to about 50–100 m. She had previously been healthy and very active. At investigation, she had no femoral pulses and a bilateral ankle brachial index of 0.6. Further evaluation with angiography showed an aortic occlusion at the level of the renal arteries, and she was reconstructed with an aorto-bi-iliac Dacron graft (16 × 8 mm) after local proximal aortic endarterectomy. The proximal anastomosis was made end to end, and the iliac end to side. Polypropylene sutures were used. The operation was somewhat technically difficult, with the proximal anastomosis having to be redone; the duration of surgery was 3.5 h, with a blood loss of about 800 ml. The immediate postoperative course was uneventful. After 3 years, the patient had distal septic microembolisation in the left leg with an abscess around the left distal graft limb. This was extirpated, the wound was drained, and a femoral-femoral cross-over graft was inserted. She was put on antibiotics for 6 months. Five years after the aortic operation, she had melaena and a decrease in haemoglobin.

Question 1

What is the time interval between aortic surgery and the presentation of an aortoenteric fistula?

A. It usually occurs in the first 48 h following aortic surgery.

B. It typically presents within the first month following the operation.

C. It may only occur in the first 5 years following the placement of the aortic synthetic graft.

D. It may present at any time during the lifetime of the patient after the placement of the synthetic aortic graft.

The patient was investigated at her primary healthcare centre with gastroscopy and colon enema, with negative results. After 2 months, she had melaena again; after further melaena 3 months later, she was referred to the hospital. On this occasion, she also had slight back pain and low-grade fewer.

Question 2

What is meant by herald bleeding?

A. Bleeding where the aetiology cannot be determined.

B. Small bleeding(s) before a large one from a major artery.

C. A "warning" bleeding before a fatal one.

D. A small haematemesis before melaena.

A gastroscopy showed a very distal duodenal "ulcer" with a green-coloured (bile-stained) graft in the bottom (Fig. 37.1). A computed tomography (CT) scan showed fluid around the proximal part of the graft, with some gas bubbles.

Question 3

How will you rule out the presence of an aortoenteric fistula?

A. Gastroscopy.

B. Computed tomography.

C. Magnetic resonance imaging.

D. Barium enema and or barium swallow and follow-through.

E. None of the above.

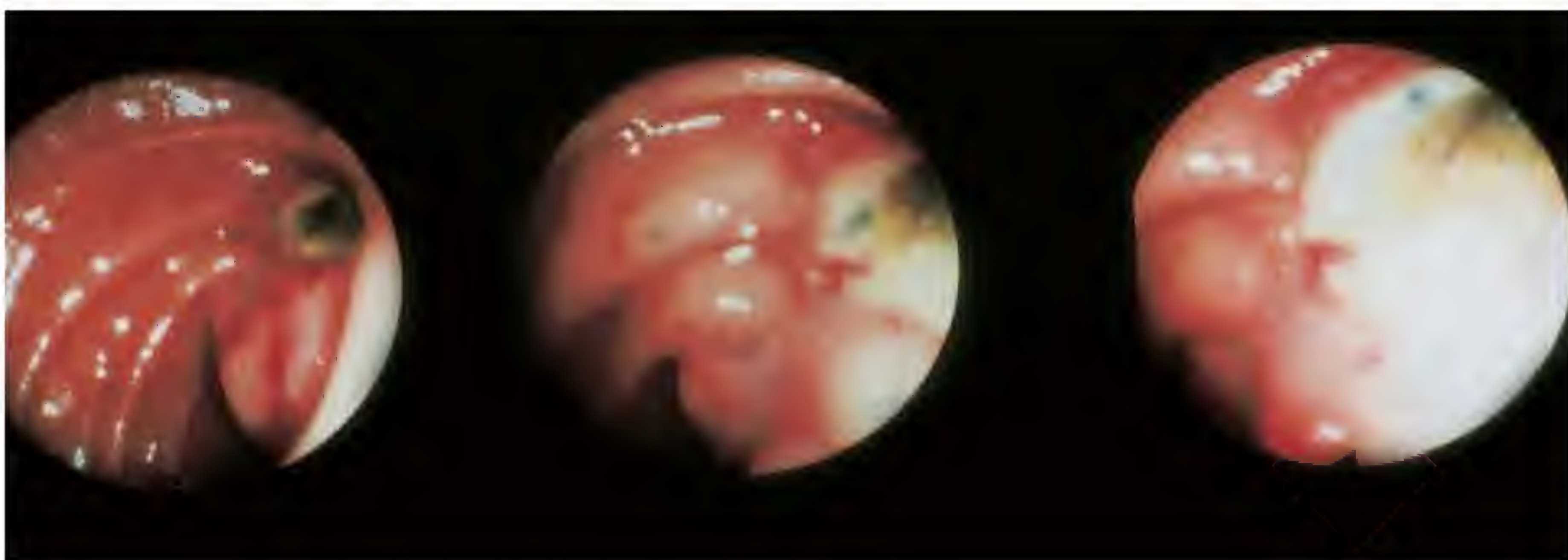


Fig. 37.1. Gastroduodenoscopy showing the Dacron graft in the bottom of an ulceration, the graft being bile stained. 🕮

Question 4

Which part of the bowel is involved in an aortoenteric fistula?

A. Duodenum.

B. Jejunum.

C. Ileum.

D. Appendix.

E. Any of the above could be involved.

Following a diagnosis of secondary aortoenteric fistula, and with the patient being circulatory stable, an axillo-bifemoral Dacron graft was inserted. During the same period of anaesthesia, the old aortic graft was extirpated. A duodenorrhaphy was made, and the aortic stump, which was about 2 cm below the renal arteries, was sutured and covered with omental tissue.

Question 5

Which treatment options are not to be recommended?

A. Stent grafting the anastomosis.

B. Wait and see if the patient starts bleeding again.

C. Extirpation of the aortic graft and then an axillofemoral reconstruction.

D. Axillofemoral reconstruction and then extirpation of the aortic graft.

E. In situ reconstruction with a new graft.

The patient recovered and she left hospital after 12 days. After 10 months, she had melaena again and was admitted to hospital. Based on her previous history, a CT scan was ordered, but she suddenly developed abdominal and back pain, large gastrointestinal bleeding – both haematemesis and melaena – and went into shock. She died before any treatment could be given. Autopsy showed a blow-out of the aortic stump with a fistula to the duodenum and also bleeding into the retroperitoneal space.

Commentary

The term "aortoenteric fistula" means a communication between the aorta and some part of the gastrointestinal tract. It is rarely primary; most often, it is seen secondary to reconstructive vascular surgery, that is, secondary aortoenteric fistula. In the majority of cases, it is seen after aortic graft insertion. It has also been reported after stent-grafting [1] and after simple aortic suture [2]. The majority (about 75 per cent) of fistulas involve the duodenum, but any part of the gastrointestinal tract may be involved. **[Q4: E]** A few patients have more than one fistula. In exceptional

cases, it can occur after other abdominal operations or radiation treatment. It is an emergency situation and should always be suspected in patients with an aortic reconstruction presenting with gastrointestinal bleeding. It can occur at any time postoperatively, which means that the patient with an aortic graft is at risk for their entire lifetime of developing a fistula. Thus, the true incidence of this condition cannot be established until all patients in a risk population have died. The longest interval reported is 18 years. Often, there is a delay of several years. **[Q1: D]** During a period of 21 years in Sweden, there are indications that the incidence has decreased to around 0.5 per cent after abdominal aortic operations [3].

Two factors have been considered to be of major aetiological importance: mechanical stress from the pulsating graft, which is in continuous contact with the intestine, and the presence of a low-grade infection. In patients with an aortoenteric fistula, there is often a history of complicated and troublesome primary graft operation or infectious complications in the postoperative course. The three most common findings at surgery are suture line contact with the bowel, pseudoaneurysm rupturing into the intestine, and graft body erosion of the intestine. To avoid complications, atraumatic surgical technique is important, avoiding bowel trauma and large haematomas. The surgeon should always try to cover the graft to avoid direct contact between the graft and the bowel.

The main symptom is gastrointestinal haemorrhage, which can range from mild melaena with anaemia to a profuse, immediately fatal haematemesis. Often, this massive bleeding is preceded by small herald bleedings, which are an important warning symptom. **[Q2: C]** About half of the patients also have septic symptoms of varying severity. In some patients, septic symptoms dominate, and the bleeding may even be occult.

There is often a long delay between onset of the symptoms and final diagnosis. In some patients with a large initial bleeding, the diagnosis is established at autopsy. The cardinal importance of a high degree of clinical suspicion for obtaining a correct diagnosis must be emphasised. Unfortunately, there is no specific diagnostic test. At gastroduodenoscopy, it is important to scrutinise the whole duodenum down to the ligament of Treitz. Observation of a bile-stained graft is obviously pathognomonic. Endoscopy is also important to reveal other sources of bleeding. CT, magnetic resonance and angiography may be helpful in showing pseudoaneurysm or fluid outside the graft, sometimes with gas in it. Conventional radiological methods for gastrointestinal examination are rarely helpful. One great problem is that the absence of abnormalities does not exclude the diagnosis. Exploratory laparotomy is indicated in patients with massive bleeding or where diagnostic efforts have been negative and the patient is still bleeding. **[Q3: E]**

The management is difficult. Total removal of all old graft material and revascularisation seems to give the best results [4]. Just closing the fistula locally always leads to recurrence and the mortality is close to 100 per cent and cannot be recommended [5]. It seems optimal to start with an extra-anatomical revascularisation of the extremities and thereafter removal of the graft. Some authors recommend a delay of a few days between the two procedures [6]; this is possible when the haemorrhage is under control. In emergency situations, an abdominal exploration with closure of the fistula and graft removal is vital, but this may lead to delayed revascularisation with profound limb ischaemia. When the graft is removed, the problem is how to deal with the aortic stump, which must be closed, preferably with double sutures. This may, however, not be possible if the distance to the renal arteries is too short. The stump is preferably covered with some vascularised tissue, and most fre-

Table 37.1. Surgical treatment options for aortoenteric fistula

Extra-anatomic bypass with resection of the infected prosthesis
Staged
Simultaneous
Resection with in situ reconstruction
Antibiotic (rifampicin)-soaked graft with omental wrap
Homograft
Autologous vein

quently an omental pedicle has been used. Some authors advocate removal of the graft and an in situ reconstruction with expanded polytetrafluoroethylene (ePTFE) graft or an antibiotic-bonded Dacron graft (often with rifampicin) [7, 8] or in situ autologous vein [2, 9] Table 37.1 summarises the treatment options for the surgical management of aortoenteric fistula. **[Q5: B]**

The prognosis is poor, with a high postoperative mortality, often several complications should the patient survive, and a risk of aortic stump blow-out, which very few patients survive. Results have improved over recent years, but aortoenteric fistula is still a very serious and challenging complication. The 5-year survival rate is 50–60 per cent [3, 7].

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38. The Optimal Conduit for Hemodialysis Access

Frank T. Padberg Jr, Robert W. Zickler and
Joseph M. Caruso

A 42-year-old type 1 diabetic of normal weight has recently progressed to chronic renal disease. Insulin-dependent diabetes mellitus (DM) has been managed by the same primary care physician for the preceding 12 years; glucose control has never been a problem in this cooperative and well-educated individual. The renal failure was initially managed with appropriate adjustments to diet and medications; the presumptive diagnosis is diabetic nephropathy. Recent laboratory tests demonstrate a creatinine of 4.1, a blood urea nitrogen of 94, a potassium of 4.8, mild proteinuria, and a creatinine clearance of 20 ml/min.

Question 1

At this juncture the physician's most appropriate course of action is:

A. Refer the individual to a surgeon for hemoaccess.

B. Refer the individual to a nephrologist to refine diagnosis and initiate specialty care. It is not time to initiate dialysis.

C. Refer the individual to a nephrologist who will refine diagnosis, and determine if there is a reversible cause for the renal insufficiency.

D. Refer the individual to a nephrologist who will evaluate the etiology of the renal insufficiency and determine if there is a reversible cause. If not, a surgeon skilled in the construction of durable hemoaccess should be consulted.

E. Refer the individual to a nephrologist to commence dialysis with a central venous catheter.

Question 2

A nephrology work-up finds no reversible cause and the patient's immune status precludes any further consideration of transplantation. The patient is referred for

construction of a hemoaccess. The most appropriate action is to perform a clinical vascular examination with specific attention to:

A. The pedal pulses and examination of the foot; extensive arterial occlusive disease is common in diabetic patients and infection would complicate any hemoaccess procedure.

B. The radial pulses and superficial venous anatomy. Book the operating room and proceed to construct an access in the upper extremity, guided by your clinical examination.

C. The radial pulses and superficial venous anatomy supplemented by a duplex ultrasound (DU) study. Book the operating room and proceed to construct an access in the upper extremity guided by these findings.

D. Immediate hemoaccess placement. Simultaneous placement of an arteriovenous fistula and a central venous catheter.

Question 3

Preoperative duplex ultrasound examination should include all *except* one of the following:

A. Both upper extremities.

B. Size and location of the arteries.

C. Location of the brachial bifurcation.

D. Assessment of the axillary and subclavian veins.

E. At least one lower extremity.

F. Size and location of the superficial veins.

G. Evaluation of the superficial veins for evidence of prior scarring.

The patient is right hand dominant. Non-invasive examination demonstrated the findings given in the caption to Fig. 38.1.

Question 4

Which procedure would be the *best* option for this individual?

A. Left brachial to basilic transposition arteriovenous fistula in the arm.

B. Right radial to basilic transposition arteriovenous fistula in the forearm.

C. Left brachial to median antecubital vein forearm loop graft (PTFE).

D. Left internal jugular tunneled, cuffed dual lumen hemodialysis catheter.

E. Left radial to cephalic arteriovenous fistula.



Fig. 38.1. Duplex ultrasound: The patient is right hand dominant. Non-invasive examination demonstrated the following findings.
Right: Cephalic (diameter 3.3 mm) and basilic (diameter 3.5 mm) veins course through both the forearm and upper arm to their junctions with the axillary and brachial veins respectively; however, both superficial forearm veins demonstrate post-thrombotic changes in the forearm. The brachial artery (diameter 4.2 mm) bifurcates into a radial (diameter 2.8 mm) and ulnar (diameter 2.7 mm) artery 3 cm below the antecubital crease; the palmar arches are intact. The deep venous structures are normal from the forearm veins through visualization of the axillary and subclavian veins.



Left: The basilic vein is post-thrombotic and thickened in the forearm; it has a normal 3.5-mm diameter lumen just below the elbow continuing into its junction with the brachial vein at mid-humerus. The cephalic vein (diameter 3.5 mm) has a normal luminal surface, is superficial, communicates with the proximal basilic at the antecubital junction, and remains patent into its junction with the axillary vein. The brachial artery (diameter 4.2 mm) bifurcates into a radial (diameter 2.8 mm) and ulnar (diameter 2.7 mm) artery at mid-humerus; the palmar arches are intact. The deep venous structures are normal from the forearm veins through visualization of the axillary and subclavian veins.

Question 5

Which of the following *best* describes when this new hemoaccess is considered mature enough to begin puncture for hemodialysis?

A. The wound is securely healed, the sutures have been removed, and there is a palpable thrill.

B. The wound is securely healed, the sutures have been removed, and there is a palpable thrill. At 2 weeks, a duplex examination demonstrates unobstructed flow, but the walls of the conduit appear to be relatively thin.

C. The wound is securely healed, the sutures have been removed, and there is a palpable thrill. At 8 weeks, a duplex examination demonstrated that there was unobstructed flow and the walls of the conduit have thickened measurably.

D. The wound is securely healed, the sutures have been removed, and there is a palpable thrill. At 6 weeks, a duplex examination demonstrates an equal volume of flow through both the fistula vein and a large branch vein at the site of the thrill.

E. Two weeks.

Your initial hemoaccess has functioned well for 6.4 years, but the hemodialysis staff has noted increasing difficulty obtaining adequate flows for the external machine circuit; arterial pressures were low at 70 mm Hg and venous pressures elevated to 350 mm Hg. You are asked to consider revision or a new hemoaccess.

A new duplex examination demonstrates progressive stenosis of the distal radial artery, and multiple sites of localized thrombosis extending into the upper arm cephalic vein. With the exception of the appropriate postoperative changes, the remainder of the examination is unchanged from that described in Fig. 38.1.

Question 6

Which is the *best* option to maintain hemodialysis?

A. Right radial to basilic transposition arteriovenous fistula in the forearm.

B. Right forearm loop graft (PTFE).

C. Left forearm loop graft (PTFE).

D. Left radial (antecubital) to basilic transposition arteriovenous fistula in the arm.

E. Left internal jugular tunneled, cuffed dual lumen hemodialysis catheter.

A new hemoaccess is constructed and an excellent thrill achieved. During initial maturation, hemodialysis is continued via the original left arm hemoaccess. Fortunately, the original left hemoaccess provides sufficient flow for adequate interval hemodialysis, but 6 weeks later has spontaneously thrombosed. Dialysis using the new hemoaccess is successful and the hemoaccess functions well for thrice weekly puncture.

Two years later you are again contacted to evaluate this individual. One year previously, an uneventful coronary bypass was performed. Subsequently, following an

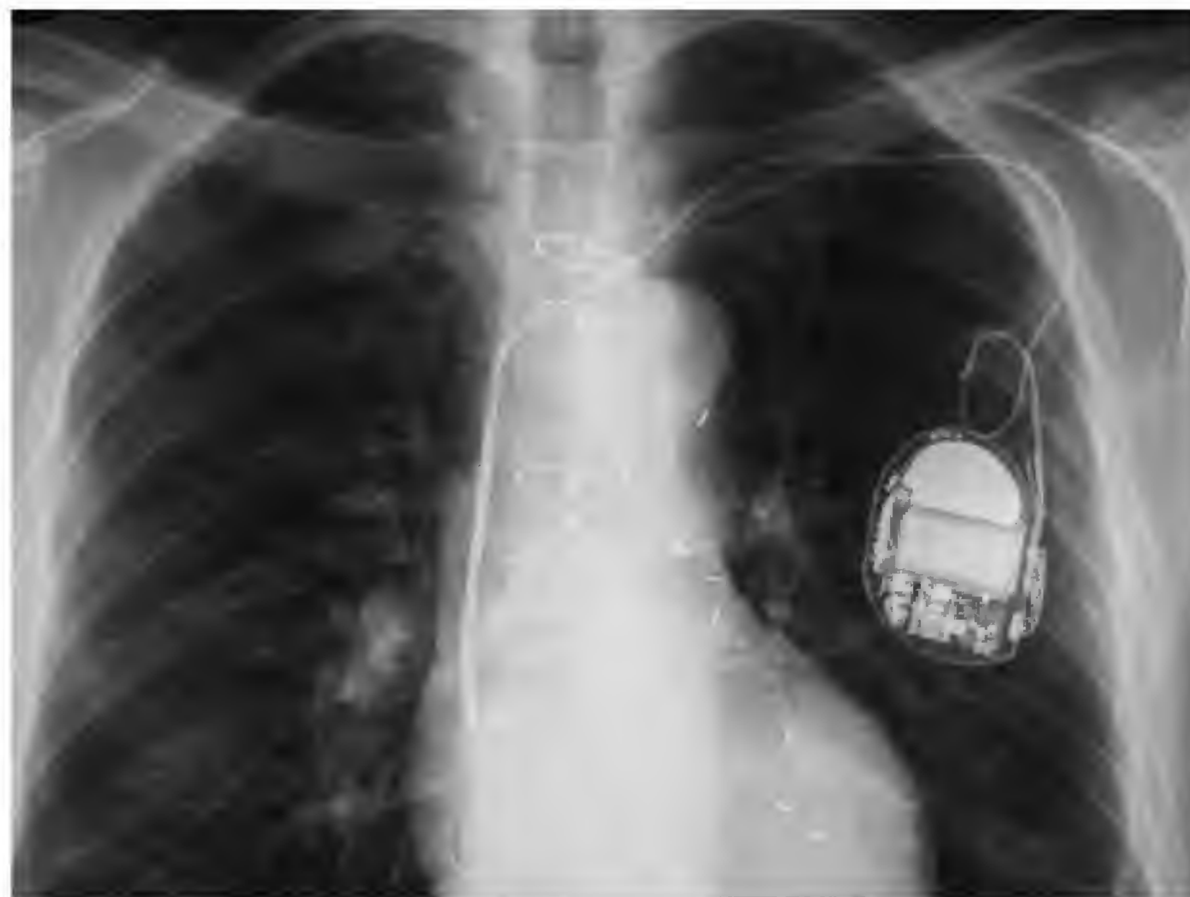


Fig. 38.2. The chest X-ray was taken at the time of the referral for arm edema.

episode of syncope, and tachyarrhythmia, a permanent defibrillator was installed on the left anterior chest wall 2 months ago (Fig. 38.2).

The patient is complaining of an obviously swollen left arm.

Question 7

What is wrong?

A. The patient's heart failure has worsened from a combination of a fixed heart rate and the increased output demanded for the hemoaccess.

B. Edema is a result of lymphatic disturbance from defibrillator implantation.

C. The patient is hypercoagulable and has thrombosis of the superior vena cava (SVC).

D. Unilateral swelling results from continuously increasing flow in the hemoaccess and enlargement of the arterial anastomosis.

E. The transvenous electrodes have induced a stenosis or obstruction of the left subclavian vein.

Question 8

The *best* treatment for this condition is?

A. Begin strong diuresis to counter the right heart failure.

B. Place the arm in a sling and elevate it to reduce the existing edema from the operation. The patient is reassured that edema following pacemaker insertion of these devices is usually self-limited and will soon resolve.

C. The defibrillator is removed and replaced in the right subclavian vein.

D. A fistulagram/venogram is performed. This will determine the etiology of the edema and may offer an opportunity for interventional therapy.

E. A hypercoagulable work-up is obtained.

All of the interventions aimed at reducing the left arm edema are unsuccessful, and the patient is discharged home. After multiple attempts, the dialysis staff reluctantly admit they are no longer able to reliably cannulate the left arm arteriovenous fistula (AVF). You are again asked to consider revision or a new hemoaccess.

A new duplex ultrasound is obtained. The appropriate postoperative findings are noted; otherwise, the relevant arm anatomy is unchanged from the initial survey as shown in Fig. 38.1.

Question 9

The optimal hemoaccess for this individual now is:

A. Right forearm loop graft (PTFE). Ligation of left hemoaccess.

B. Left internal jugular tunneled, cuffed dual lumen hemodialysis catheter. Ligation of left hemoaccess.

C. Right internal jugular tunneled, cuffed dual lumen hemodialysis catheter. Ligation of left hemoaccess.

D. Left femoral to femoral loop graft (PTFE). Ligation of left hemoaccess.

E. Left femoral tunneled, cuffed dual lumen hemodialysis catheter.

F. Right brachial-cephalic transposition.

The left arm symptoms resolve, and the new access functions well for 2.6 additional years. However, dilation begins to appear in two sites most commonly used for the tri-weekly puncture for hemodialysis. Duplex examination of the larger discerns the presence of a large pseudoaneurysm with a 0.6-mm neck communicating with the lumen of hemoaccess. The individual reports several recent episodes of prolonged difficulty achieving hemostasis after removal of the access needles. During duplex interrogation, a thrombotic plug is dislodged. Pulsatile bleeding ensues, which is controlled with 30–45 minutes of direct compression.

Question 10

The best treatment option at this time is:

A. Ligation of the hemoaccess.

B. Revision by primary closure of the pseudoaneurysm.

C. Revision with placement of an interposition, prosthetic segment.

D. Removal of the hemoaccess.

E. Continued, but close, observation of the patient with treatment initiated if the bleeding recurs.

After loss of the above hemoaccess, a new autogenous AVF was available for construction in the right arm, which remained functional until the patient's demise 3 years later.

Commentary

Question 1

The first order of business is to determine whether the individual has a reversible condition such as obstructive uropathy, drug-induced acute tubular necrosis, or another nephrotoxic condition. Commencement of hemoaccess would be unnecessary at this time since the individual has minimal symptoms; however, the degree of renal insufficiency is sufficient to predict that it will likely be required within several months to a year. Since some access procedures require several months before they are usable, an experienced surgical specialist should be contacted to construct the hemoaccess, if the nephrologist confirms that the individual has chronic progressive renal insufficiency [1]. **[Q1: D]**

Urgent or immediate hemodialysis is not indicated and because of the associated morbidity, catheter placement is contraindicated in the absence of acute renal failure. Likewise, it is inappropriate to refer directly to the surgical specialist without determining the cause of the renal insufficiency, whether it is reversible, and whether the individual should be placed on the eligibility list for transplantation. Simply referring the individual to the nephrologist is not wrong, but the best option includes the diagnostic evaluation, management of treatable etiologies, and consideration for hemoaccess assuming that commencement of hemodialysis is imminent within several months to a year. It is clear that early nephrology consultation is of benefit, from the perspective of directing appropriate therapy [2–5]. Likewise, early consideration of hemoaccess options facilitates preservation of vascular assets and reduces the incidence of catheter placement and the subsequent morbidities.

Question 2

The complete clinical vascular examination is an important adjunct to surgical planning and may direct the surgeon to either upper extremity; clearly the non-dominant upper extremity is preferred, unless a preferred access option is only available in the dominant extremity. While reliance on the clinical examination alone may be accurate in many cases, experience currently suggests that valuable information is contributed by the non-invasive ultrasound survey [6–9].

While reliable duplex ultrasound (DU) examinations may not be available in all practice situations, it does provide the "best" option by decreasing the likelihood of unsuccessful operations while increasing the options for autologous conduit. Additional information from the duplex examination may reveal proximal vein occlusion, visible superficial veins which are post-phlebitic, arterial abnormalities

(location of the brachial bifurcation, occlusive disease, inadequate palmar arch collateralization, large branch veins, and relative size of the arteries and veins).

An added reason for preoperative DU survey is that the diabetic population is the group most likely to harbor asymptomatic upper extremity arterial occlusive disease. It is unknown whether this is the reason, but functional patency of hemoaccess is usually reduced in the diabetic population [8, 10, 11]. In obese patients the lack of visible superficial veins may be countered by duplex examination; the depth of the veins precludes clinical identification and may mask prior thromboses. The depth of an otherwise acceptable vein is an important consideration and may mandate transposition to a more superficial site. Failure to identify superficial upper extremity veins on clinical examination is not an acceptable rationale for commencing access at a lower extremity site; even if no superficial veins are available, a prosthetic graft can usually be constructed between the artery and one of the deep veins in the upper extremity.

As before, there is still no indication for immediate dialysis, so that placement of a catheter at the time of the permanent access is not indicated in this clinical scenario. **[Q2: C]**

Question 3

The list includes all of the usual information needed for proper operative planning. The goal is to construct the most durable hemoaccess from autogenous tissue. When acceptable options exist only in the dominant upper extremity, it is selected; thus, both arms should be studied.

Adequate arterial inflow is essential for the fistula or graft to function properly. Failure was universal with an arterial diameter <1.6 mm in one study [9]. Although upper extremity atherosclerotic occlusive disease is uncommon in the arm, diabetics are the group most likely to have diseased arteries, and thus it should be considered for this patient. While all vascular laboratories may not subscribe to this position, no palpable pulses were described in the core scenario such that some reassurance is needed regarding adequate arterial flow.

A key function of the preoperative duplex examination is to determine the acceptability of the superficial vein network of the forearm and arm. In addition to location and diameter, identification of large branches, occluded segments, scarring, other post-thrombotic changes, and depth of vein below the skin are all critical to success. Failure was also universal when preoperative DU identified stenotic vein segments [9]. Vein diameters of less than 2.5–3 mm are generally considered unacceptable, but since there is little data to support this recommendation, the reporting standards did not incorporate a recommendation for a minimum venous diameter [7]. Complete evaluation of superficial upper extremity veins should include the forearm basilic vein [6, 12]. Transposition of the basilic vein is usually necessary whether in the forearm or upper arm, and any vein that is too deep (greater than 0.5–1.0 cm) may need to be transposed before anastomosis.

Central vein stenoses or occlusions are usually due to prior central vein catheterizations, but the surgeon should also be wary of transvenous wires from implanted pacemakers or defibrillators [1, 13, 14]. Forty percent of patients with known subclavian vein catheterization had moderate to severe subclavian vein stenoses that were clinically silent [15].

In the absence of an autogenous option, the surgeon should still be informed regarding the best location for the first graft. The anatomic variant of a high

brachial bifurcation occurs in ~10 percent of individuals. This anatomic variant may preclude placement of a prosthetic graft at a given site, but should have little adverse effect on an autogenous AVF.

Routine evaluation of the lower extremity is unnecessary, but may be considered when upper extremity sites have been completely exhausted. **[Q3: E]**

Question 4

This is a complex issue and the correct answer **[Q4: E]** is derived from a combination of experience and the recommendations of the United States Kidney Dialysis Outcomes Quality Initiative (K-DOQI). The best answer is a combination of the "best" choices summarized from the principles of all autogenous, most distal, non-dominant extremity. Thus, since almost all options are really open for this individual, the non-dominant, radiocephalic AVF is the best first choice; a potential collateral benefit is communication with (and arterialization of) the proximal basilic vein. Recognition of problems causing failure to mature are the major impediment to wider utilization of this modality [1, 8–10, 16]. Failure to mature a forearm AVF may occur in 34–53 percent, and may be less attractive in the elderly, the diabetic, and female patients [8, 9].

The proximal options of brachial and cephalic anastomoses and transpositions have experienced a higher incidence of arterial steal and ignore the basic principle of progression from distal to proximal [1]. The forearm loop graft was equated with the proximal brachial transposition by K-DOQI, but current initiatives more emphatically encourage autogenous fistula [5, 16].

Catheter access is to be avoided if at all possible, and is clearly not indicated in this situation; multiple autogenous options are available and commencement of dialysis is not emergent [4, 5, 13, 14].

A *left* radiocephalic AVF serves the dual purpose of increasing lifetime site options, and allows the distal AVF to develop more proximal veins for subsequent autogenous hemoaccess options. A forearm vein transposition has been touted as a better option than the radiocephalic AVF but this option was not available, even in the dominant extremity, because of post-thrombotic changes [12, 17].

Question 5

Although this is a common clinical question, informed decisions are difficult since there is a paucity of concrete data. There is little data to support any course of action, and the correct answer is based upon opinion [1]. **[Q5: C]** Clearly a healed wound, a palpable thrill, and unobstructed flow are essential. Two weeks is generally considered early for an AVF, and the minimum recommended interval is 6–8 weeks. For prosthetic grafts, 2–3 weeks is usually satisfactory, as long as the edema has resolved sufficiently to identify the outline of the graft. Since the functional patency is so low, a prosthetic AV graft should not be inserted until dialysis is imminent [1].

A period of 6–8 weeks is an arbitrary interval often used in practice and supported by K-DOQI. However, if at all possible, a longer interval is preferable, since a matured access is more likely to provide durable function.

A large branch vein within 5 cm of the arteriovenous anastomosis can prevent maturation, by diversion and diffusion of fistula flow and should be ligated [9].

Thickening of the wall is one of the few indicators of arterialization in the conduit walls and would therefore be desirable, but there is no data to support this course of

action, or to guide the obvious question of how thick? In reality, the progression of the individual's renal disease will likely be the best guide. If early referral can be achieved, urgent commencement of dialysis becomes a moot question and the hemoaccess is ready for use when the time arrives [1].

Question 6

The best option again emphasizes the principles of most distal site, autogenous if possible, and in this instance the recruitment of additional vein collaterals from the long-standing prior left radiocephalic AVF [17, 18]. **[Q6: D]** The initial DU examination specifically noted the communication from the cephalic feeding the basilic system; the proximal forearm basilic has a good size and does not exhibit post-thrombotic changes from this point into its termination in the brachial veins. Thus, the transposition of the proximal basilic vein, which may already be arterialized, is the preferred choice [17, 18]. The arterial inflow for this may be the proximal forearm radial artery if the vein length is satisfactory; if not the high bifurcation would still make the inflow from the radial the best choice. Even though the diameter of the artery would be less than the usual brachial diameter, this should not present a problem for construction of an autogenous fistula.

The brachial arterial variant would compromise the inflow to a left forearm loop prosthetic graft and even a brachial to axillary prosthetic graft in the arm.

The right forearm loop graft would be an acceptable alternative, except that there are good autogenous alternatives bilaterally. It moves the access to the dominant upper extremity and fails to take advantage of the previously arterialized proximal venous channels.

Any right forearm transposition is inappropriate because both veins have evidence of prior thrombosis.

Central catheter access is to be avoided if at all possible. Multiple autogenous options are available and dialysis can be maintained in the interval with the existing, but poorly functioning left radiocephalic AVF. By mobilizing a proximal basilic vein segment that has already been exposed to arterialized fistula flow, the time for maturation may be reduced [1]. The arm will need to be observed for possible arterial steal, in which case the failing AVF may need to be sacrificed and a hemoaccess catheter inserted until the new left radiobasilic transposition has matured satisfactorily [13].

Questions 7 and 8

The presence of subclavian vein electrodes in the subclavian vein is the inciting factor for subclavian vein thrombosis or stenosis whether for pacing or defibrillation. **[Q7: E]** Symptoms from acute subclavian vein thrombosis are often expressed for only a short time in patients without an arteriovenous fistula. Edema will be severely and continuously exacerbated from the additional limb blood flow of the AVF [13, 14]. Often the edema becomes chronic, and precludes accurate puncture of an ipsilateral access. Untreated venous hypertension from this combination may produce the typical symptoms of venous stasis: edema, hyperpigmentation and even ulceration. Therefore any treatment must include ligation of the access.

Although lymphatic disruption from an infraclavicular pocket incision is possible, this would be so rare as to be remarkable. The electrodes are usually inserted

indirectly and do not require surgical exposure of the vein; this reduces the likelihood of injury to the lymphatic channels in the axillary-subclavian vascular sheath. Localized swelling in the pocket would be a more likely complication than arm swelling.

There is no evidence for hypercoagulability. Since the electrodes pass through the SVC, obstruction there is theoretically possible. However, the absence of contralateral upper extremity edema or a swollen head fails to suggest SVC thrombosis. Replacing the device on the right complicates the issue immeasurably. In addition to incurring a real risk of SVC obstruction, it also places the remaining (right) upper extremity at risk for problems with subsequent hemoaccess.

The combination of a high flow AVF with probable subclavian vein obstruction suggests a rather poor prognosis for the left arm radiobasilic AVF. Thus, investigation of the etiology with a fistulagram and venogram is appropriate. **[Q8: D]** A DU should also be obtained, but since central vein visualization is poor it is inadequate to confirm the suspected diagnosis. Although unlikely, it is entirely possible that the device has nothing to do with the venous obstruction and that the outflow vein of the AVF may be stenotic from an intimal hyperplastic response in a different anatomic location more amenable to a salvage procedure. If this is a subclavian thrombosis, it may be a good opportunity to consider thrombolysis. However, even if the vein could be reopened, a subclavian vein angioplasty and/or stent has not proven a durable solution in this anatomic site [13]. Finally, thrombolysis carries a small but real risk of intracranial hemorrhage, which would be less acceptable without a real benefit. Removal of the electrodes would be complicated and risky. Unfortunately, if the obstruction is not well collateralized, the left arm should be excluded from future access options [1, 13, 14, 18].

Question 9

The best choice is the right forearm loop prosthetic graft. **[Q9: A]** Either an internal jugular (IJ) or femoral catheter site has significant clinical negatives, and fails to offer a durable solution in the face of numerous better options.

The right brachial to cephalic is an attractive option, which is considered equivalent to the loop graft in the DOQI guidelines; however, as presented in the question, ligation of the contralateral symptomatic radiobasilic AVF is not accomplished. More importantly, the two proximal transposition options remain available for construction of subsequent hemoaccess.

Ligation of the left radiobasilic AVF is essential to control the venous obstructive symptoms. While a jugular vein turn-down would offer preservation of the left radiobasilic AVF, there is not likely to be sufficient length to reach a non-thrombosed segment of axillary vein. A prosthetic extension to the jugular is another alternative. Adequate central outflow from the jugular would need to be assured by venography before further consideration of either option [14, 19].

Question 10

Although there is very little data to provide a clear answer to this clinical problem, the best choice and DOQI recommendation is prosthetic interposition. **[Q10: C]** It preserves a functioning access in someone who has already lost the use of the contralateral upper extremity to venous outflow obstruction [1, 20, 21].

Ligation solves the bleeding problem but sacrifices the access. Removal may subsequently be required, but is not essential at this juncture. Revision with primary closure of the aneurysm is unattractive since the tissues and graft material are friable and usually destroyed by the repetitive puncture. Close observation is doomed to fail with a real risk of bleeding and hemorrhage.

Although prosthetic interposition is the appropriate choice, this option is not without complications. Our own experience identified an increased incidence of infection, and good material to anastomose may require bypass of lengthy segments [22]. Recent introduction of the covered stent is an attractive, but unproven option. Percutaneous access, control of the neck of the pseudoaneurysm, and retention of hemoaccess function are currently offset by limited clinical data, and high expense [13, 23].

Comment

The initial use of distal sites, and judicious consumption of the available autogenous assets facilitated construction of several different hemoaccess sites during this patient's 14-year odyssey with hemodialysis. Problems such as these are common and require forethought and ingenuity for successful cumulative function and minimization of major complications.

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39. Acute Ischaemia of the Upper Extremity Following Graft Arteriovenous Fistula

Miltos K. Lazarides and Vasilios D. Tzilalis

A 65-year-old woman with end-stage renal disease and insulin-dependent diabetes was admitted for access construction in order to start haemodialysis. There was a lack of suitable veins to construct an arteriovenous (AV) fistula, and the patient underwent placement of a 6-mm polytetrafluoroethylene (PTFE) AV bridge graft between the brachial artery and the axillary vein in the left arm.

Question 1

Which of the following is the order of preference for placement of a permanent angioaccess in new patients requiring chronic haemodialysis?

A. (1) A brachio-cephalic AV fistula. (2) A wrist radial-cephalic AV fistula. (3) An AV PTFE bridge graft or a transposed brachial-basilic AV fistula. (4) A cuffed, tunnelled central venous catheter.

B. (1) A wrist radial-cephalic AV fistula. (2) A brachio-cephalic AV fistula. (3) An AV PTFE bridge graft or a transposed brachial-basilic AV fistula.

C. (1) A wrist radial-cephalic AV fistula. (2) A transposed brachial-basilic AV fistula. (3) A brachio-cephalic AV fistula. (4) An AV PTFE bridge graft.

Question 2

Which of the following statements represent advantages of the autologous AV fistulas over AV grafts?

A. Excellent long-term patency once established.

B. Lower complication rate.

C. Short lag time from construction to maturation.

D. Easy to correct surgically when thrombosed.

Immediately after surgery, the patient complained of numbness of the left hand with slight pain of the fingers. On examination the left radial pulse, which had existed previously, was absent, and the fingers were cold and cyanotic. Evaluation of the patient in the vascular laboratory with forearm Doppler pressure measurement revealed an index of 0.3. Interestingly the left forearm segmental pressure index was normalised after manual compression of the graft, while the left radial pulse reappeared with this manoeuvre. The evaluation confirmed an obvious haemodynamic "steal". The patient's condition deteriorated within a few hours; she developed severe, acute, painful weakness of the hand, wrist-drop, and minimal ability to flex the wrist.

Question 3

Which of the following statements regarding the incidence of steal after proximal access construction is correct?

A. The incidence of asymptomatic steal after proximal access construction, detected in the vascular laboratory, is rare.

B. Clinically obvious mild ischaemia after the construction of a proximal AV fistula occurs in about 10 per cent of cases.

C. Severe ischaemia necessitating surgical correction complicates 2–4 per cent of patients following a proximal AV fistula.

Question 4

Which of the following are indications for surgical correction of steal after proximal access construction?

A. Absence of ipsilateral preoperative existed radial pulse.

B. Severe symptoms (rest pain, paralysis, wrist-drop).

C. Abnormal forearm segmental pressure index measurement.

D. Abnormal ipsilateral nerve conduction studies.

E. Reversal of flow in the distal artery in colour-flow duplex imaging.

Urgent surgical correction was performed. Under local anaesthesia, a small segment of saphenous vein was harvested. The brachial artery was ligated just distal to the take-off of the graft. A vein bypass was constructed from the brachial artery 4–5 cm proximally to the inflow of the graft to a point distal to ligation (Fig. 39.1). Complete relief of symptoms occurred immediately postoperatively. The recovery of the patient was uneventful. She was discharged home on the third postoperative day with a palpable left radial pulse and a patent AV graft.

Question 5

Which of the following are acceptable corrective options for limb-threatening steal following proximal access construction?

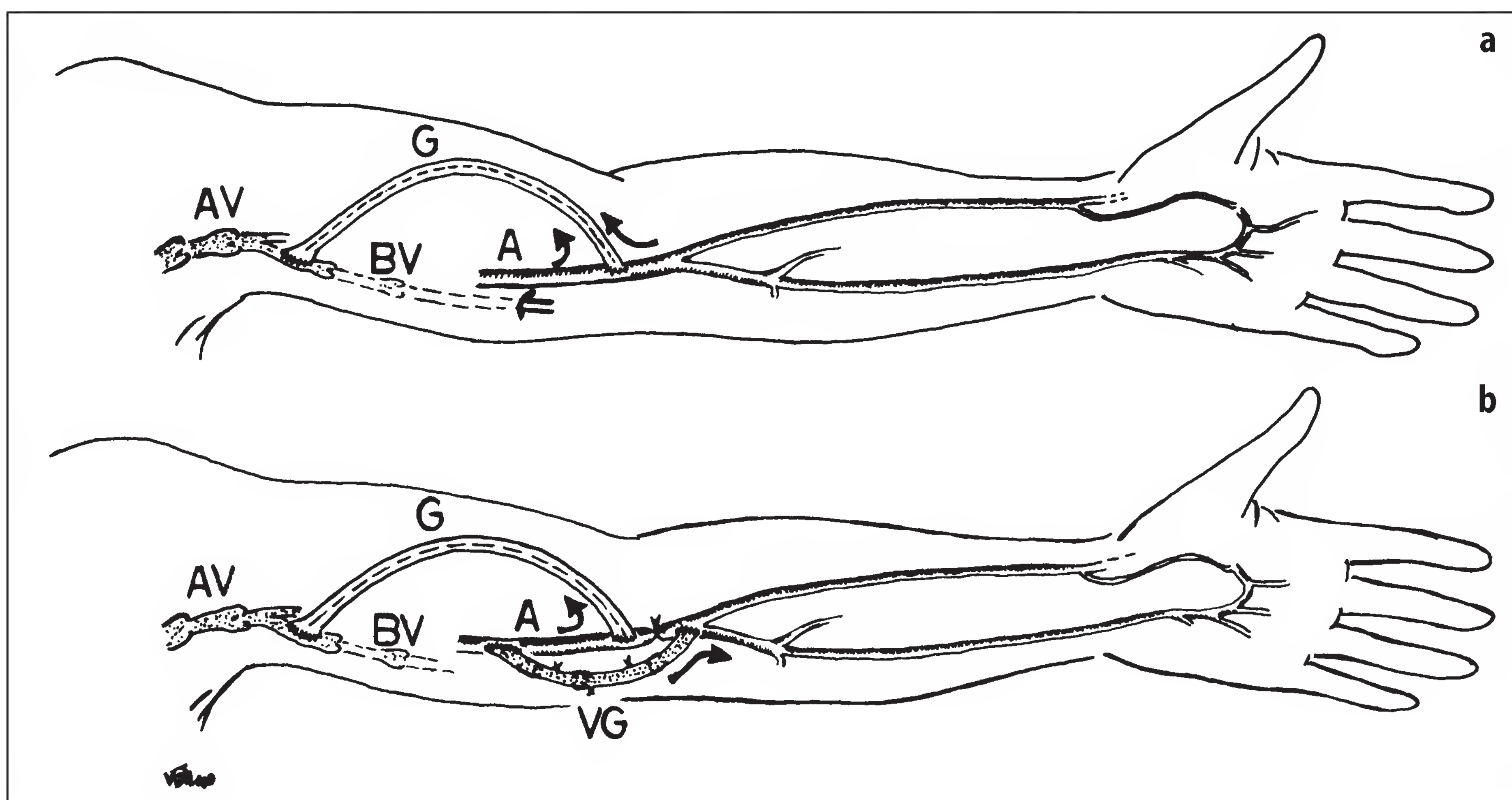


Fig. 39.1. a The operation before the creation of the corrective procedure. **b** The corrective procedure with ligation of the artery just distal to the AV graft take-off and the venous bypass from a point proximal to the inflow to a point just distal to ligation (DRIL procedure). A, brachial artery; AV, axillary vein; BV, basilic vein; G, arteriovenous PTFE graft; VG, vein graft.

A. Percutaneous transluminal angioplasty.

B. Flow reduction procedures (banding, plication or tapering of the AV fistula).

C. AV fistula closure.

D. The DRIL (distal revascularization interval ligation) procedure.

Commentary

Construction of an AV fistula provides a sufficiently superficial arterialised vein that can be punctured with ease while its flow is high enough to permit efficient dialysis. Post-dialysis compression of a matured thick-wall arterialised vein in order to stop bleeding can be obtained readily and reliably.

The classic first-choice site for an AV fistula construction is between the radial artery and the cephalic vein at the wrist, as introduced by Brescia et al. [1] in 1966. If the cephalic vein at the wrist or forearm is not usable, then the next alternative is to move to the antecubital fossa. The AV fistula can be constructed at this site between the median cubital vein and the brachial artery. The superficial cephalic vein provides enough length of vein suitable for haemodialysis venipunctures. Alternatively, if the cephalic vein is not usable, then the brachial artery can be anastomosed to the basilic vein of the upper arm. However, the latter is situated under the deep fascia in the arm, and mobilisation and transposition to a subcutaneous new position is always necessary. When an autologous AV fistula either at the wrist or the elbow cannot be created, then an AV graft using synthetic material bridging an artery and a vein in the upper extremity (either forearm or arm) is the next choice. Grafts may be placed in straight, looped or curved configurations. AV grafts and fistulas are created in the lower extremity only rarely, as they are prone to infection at this site.

The order of preference for placement of AV fistulas in patients requiring chronic haemodialysis according to dialysis outcomes quality initiative (DOQI) guidelines established by the United States Kidney Foundation is [2]:

1. A wrist radial-cephalic AV fistula.
2. An elbow (brachial-cephalic) AV fistula.

If it is not possible to establish either of these types of fistula, then access may be established using:

3. An AV graft of synthetic material (PTFE grafts are preferred over other synthetic material).
4. A transposed brachial-basilic vein fistula.

Cuffed, tunnelled central venous catheters should be discouraged as permanent vascular access. **[Q1: B]**

Recognising the superiority of the autologous AV fistulas over grafts, DOQI guidelines recommend an aggressive strategy increasing the number of native fistulas. DOQI guidelines suggest that autologous AV fistulas should be constructed in at least 50 per cent of all new patients electing to receive haemodialysis as their initial form of renal replacement therapy [2]. Bridge AV grafts should be reserved for those patients whose vein anatomy does not permit the construction of an autologous AV fistula [3, 4]. Autologous fistulas, especially distal ones at the wrist, present a lower complication rate compared with other access options [5]. A vein must be matured before use for vascular access. The time required for maturation of an autologous fistula varies among patients. It is not correct to use a fistula within the first month after its construction. Premature cannulation may result in a higher incidence of haematoma formation, with associated compression of the still soft-wall vein, leading to thrombosis. Allowing the AV fistula to mature for 3–4 months may be ideal [2].

In contrast, PTFE AV grafts need a shorter maturation time and can be used approximately 14 days after placement. Within this period, an attempt to cannulate the still oedematous arm may lead to graft laceration from inaccurate needle insertion. An AV graft may be considered matured when swelling of the subcutaneous tunnel has reduced to the point that its course is easily palpable. Additionally, after the first two weeks, fibrous tissue formation round the graft is able to seal the holes caused by each needle puncture. PTFE grafts are easily thrombectomised, with a reported unassisted patency following thrombectomy at 6 months close to 50 per cent [6]. In contrast, autologous fistulas when thrombosed are difficult to salvage [2]. **[Q2: A, B]**

The reversal of flow after creation of an AV fistula in the distal artery beyond the fistula and before the point of entry of collateral vessels has been characterised as steal. This is caused by a pronounced pressure drop in the distal artery, while pressure increases with increasing distance away from the fistula as a consequence of inflow from arterial collaterals [7]. Steal occurs in more than 90 per cent of proximal AV fistulas – when the arterial anastomosis is at the brachial artery – but in most patients, the collateral vasculature is adequate to maintain distal flow, and severe ischaemia does not develop in the hand [8, 9]. Clinically obvious mild ischaemia from steal occurs in about 10 per cent of patients. The presentation is coldness and numbness of the hand, and the symptoms resolve spontaneously

within one month [8]. The term "steal" is used inappropriately for this condition in many reports because it means reversal of flow and not any of its potential ischaemic sequelae. A wide spectrum of symptoms and signs may occur, however, such as paraesthesias and sensory loss, weakened or absent distal pulse, muscle weakness and wrist-drop, rest pain usually getting worse during dialysis, muscle atrophy and – if left untreated – digital gangrene. The reported rate of steal-induced severe ischaemia necessitating immediate surgical treatment is 2.7–4.3 per cent [8, 9]. In contrast to proximal AV fistulas, the incidence of symptomatic steal following distal radiocephalic AV fistulas is rare, at a rate of 0.25 per cent [10]. **[Q3: B, C]**

Clinical signs and symptoms of steal syndrome do not differ from those of leg ischaemia. Therefore it can be classified according to Fontaine's classification: stage I, reduced wrist-brachial pressure index, coldness of the hand or no symptoms; stage II, intermitted pain during haemodialysis; stage III, continuous ischaemic rest pain; and stage IV, ulceration and necrosis. Stages I and II should be closely observed and treated conservatively (e.g. wearing gloves) [11].

In most reports, the indication for surgical correction of steal is based on clinical grounds only [8, 9]. Low segmental pressure, as measured by Doppler, distal to the fistula is not an indication per se for surgical correction of steal. Additionally, absence of a radial pulse is a common finding in approximately one-third of patients following proximal access creation [9]. A corrective surgical procedure is indicated when proven haemodynamic steal causes severe stage III and/or stage IV ischaemic symptoms early after access construction (rest pain, paralysis, cyanosis of digits, wrist-drop). Mild ischaemic symptoms that persist beyond one month from access creation should be observed closely. When these "mild" symptoms are present for a long time, there is always a threat of irreversible neurological impairment, termed "ischaemic monomelic neuropathy". This is a serious and disabling complication causing sensorimotor dysfunction without tissue necrosis [12]. Abnormal deteriorated nerve conduction studies in the presence of even mild ischaemia are an indication for surgical correction of steal [9]. **[Q4: B, D]**

Several catheter-based and surgical techniques have been used to correct steal-induced ischaemia. Arterial stenoses proximal to the AV fistula are eligible for percutaneous transluminal angioplasty and may augment blood flow to the periphery with relief of symptoms. However, such proximal inflow stenoses contribute to steal syndrome in only 20 per cent of patients who have distal extremity ischaemia [13]. In the vast majority of cases (80 per cent) steal is caused by discordant vascular resistance and a poorly formed arterial collateral network. A variety of surgical techniques have been applied to correct limb-threatening steal including fistula closure with simple ligation, various flow reduction techniques (banding, placation or tapering) and the DRIL (distal revascularization interval ligation) procedure introduced by Schanzer et al. [14]. Ligation provides immediate improvement but requires creation of a new access. Banding–plication techniques improve distal perfusion, but it is difficult to determine the required amount of stenosis to eliminate steal while allowing a flow sufficient to sustain patency of the graft. Flow reduction procedures are attractive options in high-flow AV fistulas (>1500 ml/min) [15]. In patients with normal flow through their AV fistulas often concomitant arteriosclerotic disease causes insufficient collateral perfusion. In these cases the DRIL procedure is the treatment of choice. With the DRIL a ligature (placed distal to the take-off of the graft) eliminates the reversal of flow, while the bypass (from a point proximal to the inflow to a point just distal to ligation) re-establishes flow to the limb (Fig. 39.1). Recent reports support the efficacy of this technique [16, 17]. **[Q5: A, B, C, D]**

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40. Amputation in an Ischaemic Limb

Mohideen M. Jameel and Kingsley P. Robinson

A 74-year-old male was admitted to the hospital having sustained a crush fracture of the third lumbar vertebra while lifting a heavy box. He required opiate analgesics for the pain, which radiated to his right leg, and his mobility was severely limited. He made little progress in spite of analgesia and physiotherapy; hence, he was transferred to a geriatric rehabilitation unit, where he subsequently developed ulceration of his left heel. The ulcer was found to be suitable for community care, and as his back pain improved he was discharged home.

However, about a week later, the patient was readmitted to the general hospital with a swollen and inflamed left lower leg and foot. He was found to have a large left heel ulcer, which was 9 cm in diameter with a black necrotic base, with cellulitis of the leg and patchy ulceration of the lower leg. The arterial pulses were absent distal to the femoral pulse. No Doppler flow was detected in the posterior tibial artery, but a pressure of 90 mm Hg was found in the dorsalis paedis artery, with an ankle brachial pressure index (ABPI) of 0.4. A duplex scan excluded deep vein thrombosis. Treatment with penicillin and metronidazole resulted in some improvement. Beta Enterococcus was cultured from the ulcer.

A duplex scan of the arterial system showed little disease in the lower aorta and the iliac vessels but an atheromatous plaque with a 30 per cent stenosis in the left common femoral artery. The popliteal artery was occluded, but the anterior tibial was patent to the foot. A digital subtraction angiogram confirmed the above findings and showed that a 2-cm segment of the distal popliteal artery was patent and in communication with the anterior tibial artery, which was patent to the dorsalis paedis.

The patient's general condition was very poor. He had smoked very heavily for 40 years and had a very high alcohol intake. He was found to be unfit for major vascular reconstruction.

Question 1

Which of the following procedures may be applied in the treatment of the large heel ulcer in our patient?

A. Chemical sympathectomy.

B. Debridement.

C. Subtotal calcanectomy.

D. Free tissue transfer and microvascular anastomosis.

E. Amputation.

Debridement of the heel ulcer was carried out under epidural anaesthesia. The patient's general condition deteriorated further due to septicaemia resulting from an acute colovesical fistula, which was confirmed by the emission of gas and faecal material in the urinary catheter. His serum albumin, which was 29 g/l on admission, fell to 13 g/l. He was treated with antibiotics and nutritional support. He made slow



Fig. 40.1. Angiogram showing a functioning femoropopliteal vein bypass graft and single-vessel run-off through the anterior tibial artery.

but steady improvement but was found to have m resistance *Staphylococcus aureus* Methicilline Resistant Staphylococcus Aureus (MRSA) infection in his ulcers. About a fortnight after the debridement of his heel ulcer, his left leg became cooler and he developed rest pain in his foot.

Question 2

In what possible ways can this limb be revascularised?

Question 3

What is the role of primary amputation in an ischaemic limb?

By this time, the patient's general condition had improved somewhat to allow a left femoropopliteal bypass graft. A reversed long saphenous vein was anastomosed at the common femoral artery and was tunnelled under the deep fascia and anastomosed to the distal popliteal artery just above the trifurcation (Fig. 40.1). This resulted in good revascularisation of the foot. Following the vascular operation, a blind defunctioning transverse loop colostomy was formed.

During the initial care in the intensive care unit, the patient developed a septic episode due to chest infection and pleural effusion, which required antibiotics and aspiration. During the first few weeks, his general condition improved gradually; in particular, the sepsis in his left leg and foot made a dramatic improvement. A follow-up duplex scan showed excellent patency and function of the vein graft. The heel ulcer remained a large cavity with exposed calcaneal bone. The ankle systolic pressure was 120 mm Hg in the dorsalis paedis and 90 mm Hg in the posterior tibial artery. A further debridement of the heel ulcer was carried out, with curettage of the calcaneum under antibiotic cover.

Question 4

What is the role of subtotal calcanectomy in the healing of heel ulcers?

The possibility of a free tissue transfer and microvascular anastomosis was considered. However it was felt that the preferred approach would be to allow the area to granulate and then to apply a meshed split skin graft.2.

Although the sepsis in the leg had improved a great deal, the patient remained weak, depressed, reluctant to move, and unable to stand. There was also sloughing of the superficial part of the Achilles tendon. Therefore, an amputation became inevitable.

Question 5

Which of these amputations could be considered in this patient?

A. Chopart amputation.

B. Transtibial amputation.

C. Syme amputation.

D. Transfemoral amputation.

E. Lisfranc amputation.

Question 6

Which of the following is/are likely to predict good healing of a transtibial amputation?

A. Patent profunda femoris artery.

B. Thigh doppler pressure greater than 70 mm Hg.

C. Patent segment of popliteal artery.

D. Transcutaneous PO2 measurement of 20 mm Hg.

E. Skin blood flow levels greater than 2.5 ml/100 g/min.

A transtibial level was considered, but it was feared that amputation at this level would result in thrombosis in the saphenous vein graft due to the loss of run-off. Therefore, amputation at a more distal level was preferred, and hence a further angiogram was performed to assess the vascularity of the dorsal flap, which showed good patency of the dorsalis paedis artery. Consent was obtained for a transtibial amputation, but the possibility of a more distal procedure was included. A trial dissection was made for a dorsal flap transmalleolar amputation [1]. The instep skin was found to be very well vascularised, and the dorsal flap was raised, allowing disarticulation of the ankle joint and division of the Achilles tendon and the malleoli (Fig. 40.2). The articular surfaces at the lower ends of the tibia and fibula were removed, and the long dorsal flap was reflected posteriorly and sutured loosely. The lower leg ulcers were cleaned and dressed in preparation for skin grafting. With relief from pain and sepsis, the patient made a progressive recovery. The presence of *Pseudomonas* organisms was a contra-indication to skin grafting, and hence priority was given to the patient's rehabilitation. He was again transferred to a geriatric unit for rehabilitation.

Question 7

What factors may delay the healing of an amputation stump?

The perfusion of the residual limb had remained satisfactory and the lower leg ulcers showed accelerated granulation and epithelialisation. The ankle stump had healed soundly with stable tissue on which the patient could stand (Fig. 40.3). He has been fitted with a prosthesis, consisting of a flexible foot, multiaxial ankle joint, and a socket extending to the patellar tendon and tibial condyles to take some of the weight-bearing load. However, the patient can now stand without the prosthesis and can walk with it using a walking frame. He requires further physiotherapy to correct flexion contracture of his knee, which developed due to prolonged hospitalisation.



Fig. 40.2. *(left)* Leg before amputation, showing line of incision of the dorsal flap and loss of most of the heel pad. 📖

Fig. 40.3. *(right)* Well-healed stump. 📖

Commentary

Primary amputation as a treatment for the critically ischaemic limb has been made unnecessary by the progress of vascular surgery. The only indication for primary amputation in ischaemic disease occurs when the extent of irreversible gangrene is such that a functional extremity will not be produced by a revascularisation procedure. Such cases are occasionally encountered where the major part of the foot or the lower leg has become gangrenous. The assessment of the foot skin microcirculation has been shown to be useful in predicting imminent amputation [2]. Whether it is justified to perform a revascularisation procedure in order to obtain a more distal level of amputation is controversial, the problem being that failure of the revascularisation procedure will inevitably require a higher amputation level. It is, however, fully justified to strive for an amputation at the most distal level that can be expected to heal without delay or late breakdown, avoiding the need for a second surgical procedure, whether debridement, resuture or re-amputation. **[Q3]**

It is widely reported that revascularisation procedures such as thrombolysis, distal bypass and balloon angioplasty with or without stent insertion, and in some cases a combination of these procedures, have all resulted in a measurable fall in

the rate of major amputations [3, 4]. Although the evidence is conflicting, it seems that there is no compelling evidence that the failure of such a procedure will require a higher level of amputation than if a primary amputation had been carried out in the first place [5, 6]. It is possible that while a graft is functioning, in the time that has elapsed some collateral development may result in a better situation when the graft fails than at the time when it was first placed in position. **[Q2] [Q1: B, C, D, E]**

One difficulty that has emerged from this encouraging process is the decision of what constitutes a worthwhile residual limb if some part has to be removed at the time of the limb salvage procedure. There is no doubt that the amputation of one or all toes at the time of revascularisation is amply justified by the rapid healing and the satisfactory function that results. However, when larger parts of the foot require removal, then the functional outcome becomes less predictable, particularly so in diabetic patients who represent a different group although sharing some of the same problems. The loss of a digit and metatarsal in a "ray" type of amputation may be minimal for the fourth or fifth ray in the foot. The removal of central rays may result in very slow healing, and the removal of the hallux and first ray has a marked effect on walking, despite skilled podiatric attention. The transmetatarsal amputation may heal after revascularisation, and the gait that results with a filled shoe is equivalent to that obtained with a transtibial prosthesis, while retaining the advantages of a living limb that allows normal standing on barefoot.

However, the classical Lisfranc operation is much less satisfactory. It involves disarticulation of all the metatarsals at their base, and the residual cuboid and cuneiforms require a large area of skin to give satisfactory cover; the modified shoe and resulted gait are not very satisfactory. Chopart amputation, in which the cuboid and cuneiform are resected, is even less satisfactory, with an inherent tendency to plantar flex and evert the foot where gait problems are even more severe. While these can be corrected by tendon transplantation, this is an unsatisfactory option in the vascular patient. Amputation at the ankle has had a limited place in the management of vascular patients; this is a technically difficult operation, as the blood supply to the soft tissue heel flap is tenuous in the healthy patient and may be compromised in the patient with vascular disease. The whole heel flap depends on the calcaneal branches of the posterior tibial and peroneal artery. These run close to the malleoli and, if not damaged during surgery, are compromised by the anterior folding of the heel flap to complete the operation. A further limitation is the frequency of disease or damage of the skin of the heel itself, as occurred in our patient. Here, the whole of the soft tissue of the heel and some of the underlying calcaneum were necrotic and infected, which would exclude any ankle disarticulation of the traditional Syme type. However, in our patient, the arterial perfusion of the dorsum of the foot was adequate for healing. Hence, a myocutaneous flap from the dorsum of the foot was utilised to cover the modified bone end of the tibia and fibula, after disarticulation and removal of the foot, as reported previously [1]. By resecting the malleoli and cartilaginous surface of the tibio-talo fibular articulation, a flat bone surface is produced. The anterior flap readily covers this and can be attached to the posterior skin, while the Achilles tendon is fixed to the periosteum. This arrangement will produce an extremity similar to that of Symes operation, in which the heel flap is preserved but is a few centimetres shorter. While still allowing ground contact, this stump permits a more satisfactory prosthesis to be used. While both Syme and dorsal flap trans-

malleolar amputation can take the body weight for a few steps without any cover, for natural walking a patella tendon and tibial condyle weight-bearing prosthesis is required. It seems that, psychologically, to lose a foot is less traumatic than to lose half a leg. **[Q5: B, D]**

It is claimed that for critical ischaemia, certainly at the time before distal bypass was frequently used, the transtibial amputation was applicable to most situations. This was the best major amputation level for the patient, as it allowed a short prosthesis, was a simple fixation, was lightweight and cosmetically acceptable, and, for elderly patients, was easily applied and removed. For the younger patient, it provides a near-normal gait and the ability to run. To avoid skin breakdown over the sharp tibial border, and to make a satisfactory fit in the standard California patella tendon-bearing socket, the stump and the bone ends must be shaped carefully; the surgical technique is very important to obtain good function and satisfactory prosthetic fitting. While the long posterior flap technique of Kendrick, Burgess and Romano may achieve these objectives, the skew sagittal flap is matched more precisely to the requirements.

Whatever technique is used, the criteria for wound healing and absence of pain depend on an adequate perfusion available at the level. A huge amount of work worldwide has endeavoured to define the best technique for determining this all-important factor. Of the many procedures that are available, the best is clinical guide providing that the amputation is decisively above the area of rest pain, the skin has normal warmth, colour and sensation, and muscle function is unimpaired. The transcutaneous oxygen level must exceed 40 mm Hg. The popliteal systolic pressure must be greater than 50 mm Hg, with an increasing risk of impaired healing and pain as the value diminishes, and healing unlikely to be achieved at all at a value of less than 20 mm Hg. Cutaneous laser Doppler studies are helpful, and emission thermography may give valuable information for planning the best type of skin flap for the individual patient. For a detailed review of the available methods, the reader is referred to Sarin et al. [7] and Clyne [8]. A similar assessment can be applied to transmalleolar or the transfemoral amputation. **[Q6: A, B, C, E]**

The knee disarticulation has a controversial place in that the amount of skin required to cover the femoral condyles is only a little less than is required for a transtibial amputation. However, the skin around the knee has a good blood supply; provided this is not compromised by outside pressure or badly designed resulting in tight skin flaps, then the soft tissue cover of the condyles is not a problem, and as an amputation that requires no bone cutting, it is simple, quick, quiet and atraumatic. It has a good reputation for absence of postoperative pain, and the prosthesis is simplified as the stump is end bearing and patients can kneel on the stump.

The Gritti–Stokes amputation at a supracondylar level is modified by the retention of the patella, which is united to the bone end. With a similar reputation for healing and lack of trauma, this has enthusiastic support in some centres. Occasionally, it is painful due to non-union of the patella and femur, which limits prosthetic use. The transfemoral amputation, which must be 10–12 cm above the knee joint line, is often needed for the severe and critically ischaemic limb [9]. For optimum function, the muscles must be attached to the bone by myodesis, and opposing groups to each other by myoplasty. The bone end must be rounded carefully with sufficient soft tissue to cover it comfortably. Soft tissue retraction frequently occurs and may even lead to protrusion of the bone through the skin, with a

great deal of pain. In extreme instances of aortic or common iliac occlusion, tissue necrosis may demand a hip disarticulation, which can usually be performed without difficulty, although the inherent severity of the disease brings a high morbidity and mortality. In the upper limb, the level of amputation is determined more easily on clinical grounds, and prosthetic advice should be obtained where possible before the amputation to secure the optimum level and type of amputation for the available prosthesis.

In our patient, the conflict from the amputation point of view was between a transtibial amputation, which would be decisive and lead to acceptable prosthetic use at an early stage without the need for healing, but which might have compromised the bypass graft and required revision. It was therefore decided to proceed to the distal amputation, accepting that time would be required to heal the ulcerated skin in the lower leg following revascularisation. This has had a penalty of hospitalisation time and illustrates the many factors that may lead to the selection of a particular amputation level.

The vascular amputee, by nature of the extent and severity of their atheromatous disease, has been shown to have a diminished life expectation and an appreciable risk of a further amputation of the contralateral limb [10]. Therefore, there is considerable emphasis on utilising the patient's remaining lifespan to achieve a reasonable quality of life – if possible, independence – in their normal environment. This objective may not be achieved if there is delayed healing of the amputation stump, or problems with the amputation stump, which delay prosthetic fitting and use. There may be factors that cannot be avoided that lead to the patient needing a wheelchair or institutional care [11]. It is particularly important that the amputation level selection is accurate as the principal cause of delayed healing or breakdown of an amputation stump is inadequate blood supply. This will result in the stump being painful and prone to infection, which may precipitate the breakdown. **[Q7]** More general factors may contribute, in particular tobacco smoking and nutritional deficiency with a low total protein and albumin level, as in our patient [12]. The presence of renal insufficiency, liver dysfunction, advanced age and poorly controlled diabetes could contribute to wound breakdown in the amputation stump. Whatever the cause, the need for secondary surgery, in particular re-amputation, will contribute significantly to the morbidity and mortality of the procedure, delay the rehabilitation of the patient, and prejudice the degree of rehabilitation that the patient achieves. A further factor that is particularly difficult to assess but that is often significant in limiting the ability of the patients to use the prosthesis is their cognitive state and ability to learn. One advantage of management by a multidisciplinary team is that the patient who is unable to use a prosthesis will be identified at an early stage and rehabilitated to wheelchair independence. If this selection is not made, then the provision of prosthesis and a failure to use it will be a disappointment to the patient at considerably unnecessary expense [13].

With this exception, a positive and enthusiastic attitude of a multidisciplinary team to the amputee suffering from vascular disease should make the patient feel that the turning point has been reached and a return to independent activity is now possible. The amputation is not a failure but a means to restoration of function.

Amputation is now performed less frequently by the vascular surgeon [3, 4]. The improvement in limb salvage is diminishing the practical experience of amputation technique. It is more important now than ever before that the surgeon realises that

amputation is not just cutting off the diseased part, but also making an amputation stump that the patient will rely on for the rest of their life so that they can expect optimum function [14]. The vascular surgeon must therefore be fully competent to produce an amputation stump that will function well with the available prosthesis and will allow them to return to an independent way of life without unnecessary delay after the operation.

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41. Congenital Vascular Malformation

Byung-Boong Lee

A 10-year-old girl presented with a history of recurrent painful swelling of the left knee with mild ecchymosis. The latest episode of tender swelling of soft tissue along the left knee was preceded by a direct blow to the area during a ball game. In addition, she has had an abnormally grown left lower limb with scattered multiple soft tissue masses throughout the limb since birth.

Physical examination revealed diffuse swelling of the entire left limb, which was longer and larger than the opposite limb and more pronounced along the foot and lower leg. The swollen limb had slightly increased firmness on palpation throughout its entire length except for the soft tissue mass areas.

Multiple soft tissue masses were easily compressible and scattered from the dorsum of foot to the upper thigh; their diameters varied between 2 and 8 cm.

Similar lesions were also noticed at the left perineum, left labia, left lower abdomen, and left flank. Diffuse swelling along the medial side of left foot collapsed spontaneously when the foot was elevated.

Further evaluation of the skeletal system revealed the left lower extremity to be 5.0 cm longer – 3.0 cm longer in the tibia and 2.0 cm longer in the femur – in total length than the right lower extremity, accompanied by pelvic tilt and compensatory scoliosis of the lower spine.

However, the patient had minimal limitation of her daily activities except for moderate limping.

Family history and past history were unremarkable except for a vague history of cellulitis along the affected limb.

Question 1

What is the most fundamental problem on which clinician should focus in order to establish the proper diagnosis and treatment of this condition?

A. Scoliosis with pelvic tilt.

B. Abnormal long-bone growth with length discrepancy.

C. Abnormal swelling of lower limb with scattered soft tissue tumors.

D. Mechanical problem of knee joint with symptoms.

Question 2

What is the most basic laboratory test required to verify the nature of the problem?

A. Lumbosacral spine assessment.

B. Radiologic assessment of bone length discrepancy.

C. Duplex ultrasonography for the hemodynamic assessment.

D. Locomotive test including gait evaluation.

Question 3

Which of the following non-invasive studies could be most useful in the clinical diagnosis of the disease complex in our patient?

A. Volumetric assessment of limb size.

B. Special radiologic study of epiphyseal plate of abnormally long bone.

C. Magnetic resonance imaging (MRI) study of soft tissue masses.

D. Transarterial lung perfusion scintigraphy.

E. Bone scan.

Question 4

Which of the following non-invasive tests is not appropriate to assist in the differential diagnosis for the extremity lesions in our patient?

A. Whole-body blood-pool scintigraphy (WBBPS).

B. Computed tomography (CT) scan.

C. Radionuclide lymphoscintigraphy.

D. Transarterial lung perfusion scintigraphy (TLPS).

E. Lymphangiography (lymphography).

Clinical Evaluation

This patient underwent a thorough investigation of the nature and extent of the congenital vascular malformation (CVM) involved.

A combination of various non- to minimally- invasive studies were performed to confirm the clinical impression of venolymphatic malformation (VLM): duplex

ultrasonography, whole-body blood-pool scintigraphy (WBBPS), magnetic resonance image (MRI) study, transarterial lung perfusion scintigraphy (TLPS), and/or radionuclide lymphoscintigraphy.

The primary hemodynamic impact and the secondary musculoskeletal impact of the venous malformation (VM) were assessed as the main CVM lesion in addition to the extent/degree of each component of the VM, truncular (T) and extratruncular (ET) form, involved in the extremity.

A thorough skeletal evaluation of the long-bone growth discrepancy of the lower extremity and the degree of pelvic tilt with its compensatory scoliosis was also made with conventional bone X-rays.

The TLPS assessment was performed substituting arteriographic investigation of the lower extremity for the possible hidden micro-arteriovenous malformation (AVM) lesion, which was marginally indicated due to an unusually increased venous flow by the isolated VM lesion alone on the duplex scan under the normally developed and functioning deep vein system.

An ascending phlebography was also performed together with the percutaneous direct-puncture phlebography as a therapeutic guide; mandatory confirmation of the presence of a normal deep vein system of the lower extremity was made before starting the treatment to the infiltrating ET-form lesion of the VM.

The final diagnosis confirmed extensive involvement of the VM as an infiltrating type of the ET form causing serious clinical impact directly to the venous system hemodynamically as well as to the skeletal system to induce abnormal long-bone growth of the left lower extremity. A moderate degree of venectasia as a T form of VM along the left femoral-popliteal vein segment was also found, by WBBPS, MRI and duplex scan, and subsequently confirmed by separate ascending phlebography.

A venectasia of the femoral vein was assessed to have a limited clinical significance at this stage in comparison to the ET-form lesions of the VM.

The lymphatic malformation (LM) component which is mixed with the ET form of VM, was confirmed as the ET form, giving minimum and limited clinical impact so that a conservative management/observation was instituted for this LM component.

Therefore, the ET-form lesions of VM along the knee region were selected for active treatment as a priority; this was followed by the ankle and foot lesions.

The primary indication to initiate the treatment immediately was that these lesions were potentially limb-threatening (e.g. hemarthrosis) due to their proximity to the joints with increased vulnerability to repeated trauma, especially as a cause of her knee symptoms.

The treatment was further indicated to arrest/slow down their impact on abnormal long-bone growth.

Multiple infiltrating ET lesions of the VM along the knee region, which is surgically not amenable, were selected for ethanol sclerotherapy as independent therapy. Multisession ethanol sclerotherapy was given using 100–80 per cent absolute ethanol in calculated dosage – not exceeding 1.0 mg per kg of body weight as maximum dose per session – by direct puncture technique under general anesthesia. Close cardiopulmonary monitoring during the procedure was ensured to control and/or prevent transient pulmonary hypertension by the unavoidable spillage of ethanol into the systemic circulation from the lesion during treatment.

The symptomatic lesions along the knee with recurrent painful swelling following minor injuries were controlled well without complication/morbidity and substan-

tially reduced the risk of intra-articular bleeding and subsequent hemarthrosis. Subsequently, the ET-form VM lesions at the foot and ankle underwent surgical excision following preoperative multisession ethanol and *N*-butyl cyanoacrylic glue embolosclerotherapy with much reduced perioperative morbidity to improve foot function.

Following successful control of multiple VM lesions along the knee, ankle, and foot with priority as a potentially limb-threatening condition, other VM lesions, scattered throughout the lower extremity, were also treated with absolute ethanol to assist further attempts to arrest the abnormal long-bone growth of the lower extremity. The abnormal long-bone growth is attributed to these VM lesions scattered within the muscular structure of the lower extremity in the extensive infiltrating type of ET, with significant impact on the venous circulation along the epiphyseal plate.

In addition to the multisession embolosclerotherapy as independent and/or adjunct perioperative therapy to the VM lesions, the conservative supportive measures to improve and/or maintain overall venous function have been supplemented with the use of a graded compression above-knee stocking to prevent chronic venous insufficiency.

The final decision for the T-form lesion was left femoral-popliteal venectasia, but it was decided to defer treatment until urgent treatment of the ET form of the VM was finished, but to keep it under close observation. It might eventually require treatment (e.g. venorrhaphy, venous bypass) to prevent development of venous thromboembolism when significant venous flow/volume reduction should occur following successful control of the ET form of VM lesions. The hemodynamic consequences of the treatment of such extensive ET-form lesions directly affect total venous blood volume through the deep vein system.

The LM component in this patient was treated only with complex decongestive therapy (CDT) in order to prevent full development of lymphedema. The infiltrating ET form of LM detected together with the ET form of VM has been shown to put extra burden on the marginally normal lymph-conducting system on lymphoscintigraphic evaluation. Therefore, continuous surveillance for aggressive preventive measurement of local to systemic cellulitis along this ET form of LM lesions is mandated.

This patient will continue to be managed by the multidisciplinary team of the CVM Clinic at regular intervals for her entire lifetime, through periodical follow-up assessment of the treatment results and the natural course of the untreated lesions.

Question 5

What is the first priority in the management of this patient?

A. Correction of scoliosis.

B. Correction of bone length discrepancy.

C. Control of abnormal hemodynamic status of lower extremity by vascular lesions.

D. Correction of gait with physical therapy and shoe adjustment.

E. Biopsy of the soft tissue mass.

Question 6

Which of the following is not an indication for the treatment of venous malformation?

A. Lesion located near to the limb threatening region.

B. Life threatening lesion.

C. Symptomatic lesion.

D. Lesion with complication.

E. All the lesions regardless of their condition.

Question 7

What is the International Society for the Study of Vascular Anomaly (ISSVA) recommended and most popular strategy with respect to limb length discrepancy?

A. Immediate surgical intervention to the epiphyseal plate to arrest further abnormal growth of the affected bone.

B. Conservative treatment of limb length discrepancy only with physical therapy and shoe adjustment.

C. Hemodynamic control of venous malformation as a priority whenever possible.

D. Corrective surgery of bone for length discrepancy with the unaffected limb as a priority.

E. None of the above.

Question 8

What is the current trend of therapeutic strategy for venous malformation lesions in the lower extremity?

A. Surgical excision of the vascular lesions and related procedure only.

B. Transarterial embolotherapy only.

C. Transvenous sclerotherapy only.

D. Multidisciplinary approach with surgical therapy and embolosclerotherapy.

E. Percutaneous direct puncture sclerotherapy only.

Question 9

What is the general consensus on invasive investigations (e.g. arteriography; phlebography) for venous malformation?

A. There is no indication for invasive investigation for the diagnosis and treatment of venous malformation.

B. Invasive investigations are indicated in every suspected case of venous malformation for the confirmation of the diagnosis.

C. Invasive investigation can be reserved for the therapeutic regimen as a road map and/or occasional differential diagnosis.

D. Invasive investigation should be used only for the follow-up assessment.

E. None of the above.

Question 10

What is the most important precondition for the treatment of venous malformation in the lower extremity?

A. History of deep vein thrombosis.

B. Combined lymphatic malformation.

C. Vascular-bone syndrome: length discrepancy of the long bone.

D. Existence of deep vein system.

E. Skin lesion with ulcer and necrosis.

Question 11

What has to be included in the differential diagnosis of venous malformation?

A. Lymphatic malformation.

B. AV shunting malformation.

C. Infantile hemangioma.

D. Capillary malformation.

E. All of the above.

Commentary

Congenital vascular malformation (CVM) is regarded as one of the most difficult diagnostic and therapeutic enigmas in the practice of medicine. Vascular surgeons often take this vascular malformation quite casually without any specific knowledge, and this cavalier approach can end in failure. Clinical presentations of the CVMs are extremely variable, ranging from an asymptomatic birthmark to a life-threatening condition. This variability in the clinical presentation has been a major challenge to even the most experienced clinicians [1, 2]. Many attempts to control this ever-challenging problem, especially in the twentieth century, were led by surgeons alone, but with mostly disastrous results because of poorly planned and over-aggressive surgical treatment carried out on the basis of limited knowledge [3, 4]. Recently, a multidisciplinary approach was introduced with a new concept based on

Table 41.1. Hamburg classification of congenital vascular malformation: 1988 consensus with modification

Species	Anatomical form
Predominantly: Arterial defects	Truncular forms: Aplasia or obstruction Dilatation Extratruncular forms: Infiltrating Limited
Predominantly: Venous defects	Truncular forms: Aplasia or obstruction Dilatation Extratruncular forms: Infiltrating Limited
Predominantly: Arteriovenous (AV) shunting defects	Truncular forms: Deep AV fistula Superficial AV fistula Extratruncular forms Infiltrating Limited
Combined: Vascular defects	Truncular forms: Arterial and venous Hemolymphatic Extratruncular forms Infiltrating hemolymphatic Limited hemolymphatic
Predominantly: Lymphatic defects	Truncular forms: Aplasia or obstruction Dilatation Extratruncular forms: Infiltrating Limited

Hamburg classification [5, 6]. The Hamburg classification gives excellent clinical applicability with minimum confusion because the new terminology itself provides substantial information on the anatomico-pathophysiological status of vascular malformation; it has become the most fundamental rationale for the advanced concept of vascular malformation [7–9] (Table 41.1). It classifies complex groups of various vascular malformations based on the predominant type: VM, LM, AVM, and combined form which is mostly hemolymphatic malformation (HLM). The VM is the most common type of CVM together with LM and they often combine together to make the clinical condition quite complicated.

When this HLM consists of only two components, that is, VM and LM, it is grouped separately as VLM which is almost equivalent to Klippel–Trenaunay syndrome, where our patient belongs.

The new Hamburg classification provides critical information relating to recurrence based on precise information of embryonal stage when the developmental arrest has occurred [9, 10].

When this developmental arrest occurs in an early stage of embryonal life, it remains with mesenchymal cell characteristics so it is grouped as ET form; when it occurs in the later stage of embryogenesis, it is grouped as T form with lack of mesenchymal cell characteristics, which is extremely crucial for the clinical management.

This patient presented with the most common clinical manifestation of CVM, with various findings related to the venous malformation (VM) as primary lesion as well as its secondary phenomena since birth (Fig. 41.1). Among many clinical findings, this patient presented with multiple, scattered, soft tissue mass lesions along the lower extremity, extending from the toe to flank, which provide the necessary clues to initiate proper investigation of VM as the etiology of this condition [11, 12]. **[Q1: C]**

Relatively firm diffuse swelling of the entire left lower extremity, in addition to the abnormal long-bone growth with length discrepancy, may give further clues to the investigation on the combined nature of VM and LM as the cause of the vascular-bone syndrome [13, 14].

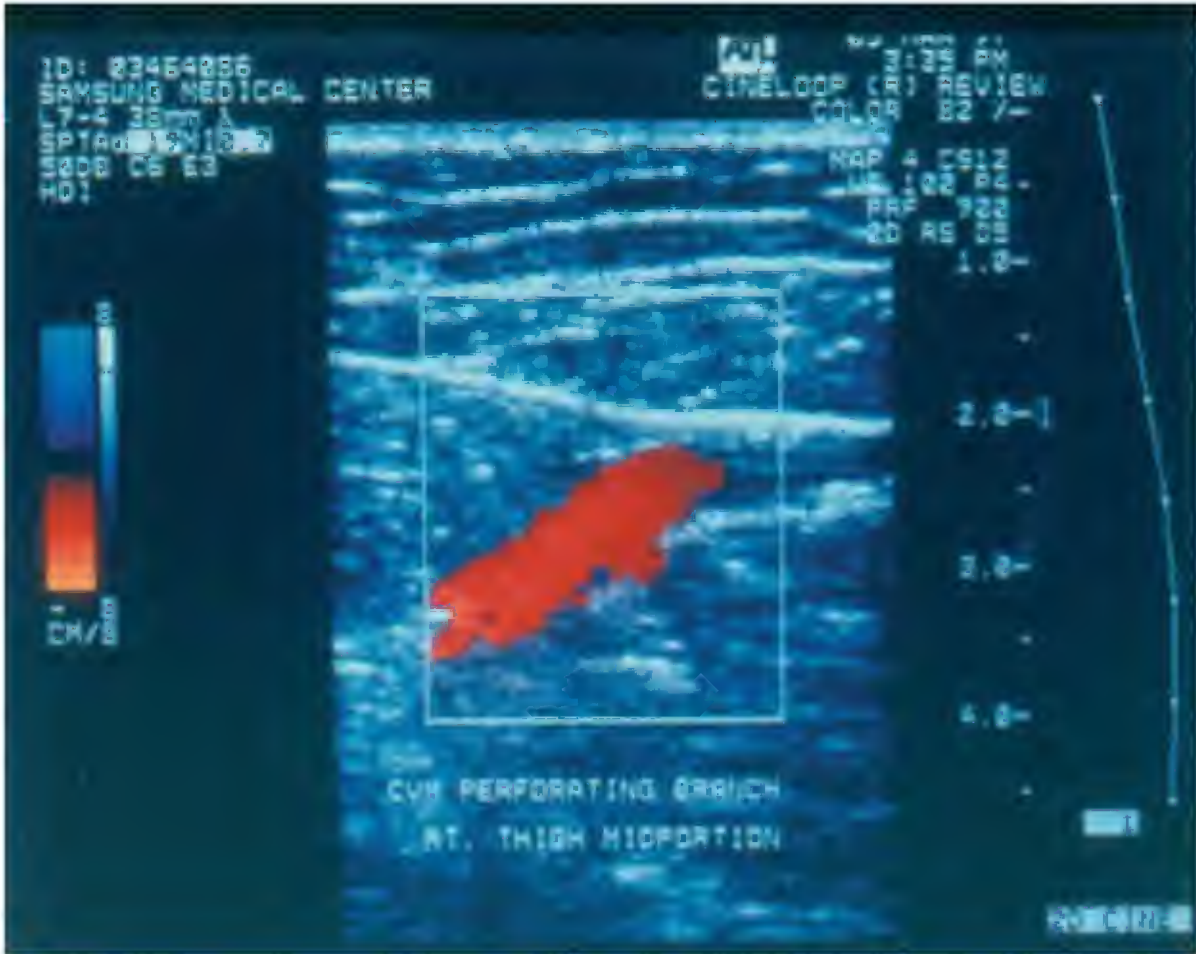


Fig. 41.1. Clinical appearance of the patient, with extensive VM lesions scattered along the left lower extremity from toe to thigh, with extension to the perineum, labia, lower abdomen and flank, left.

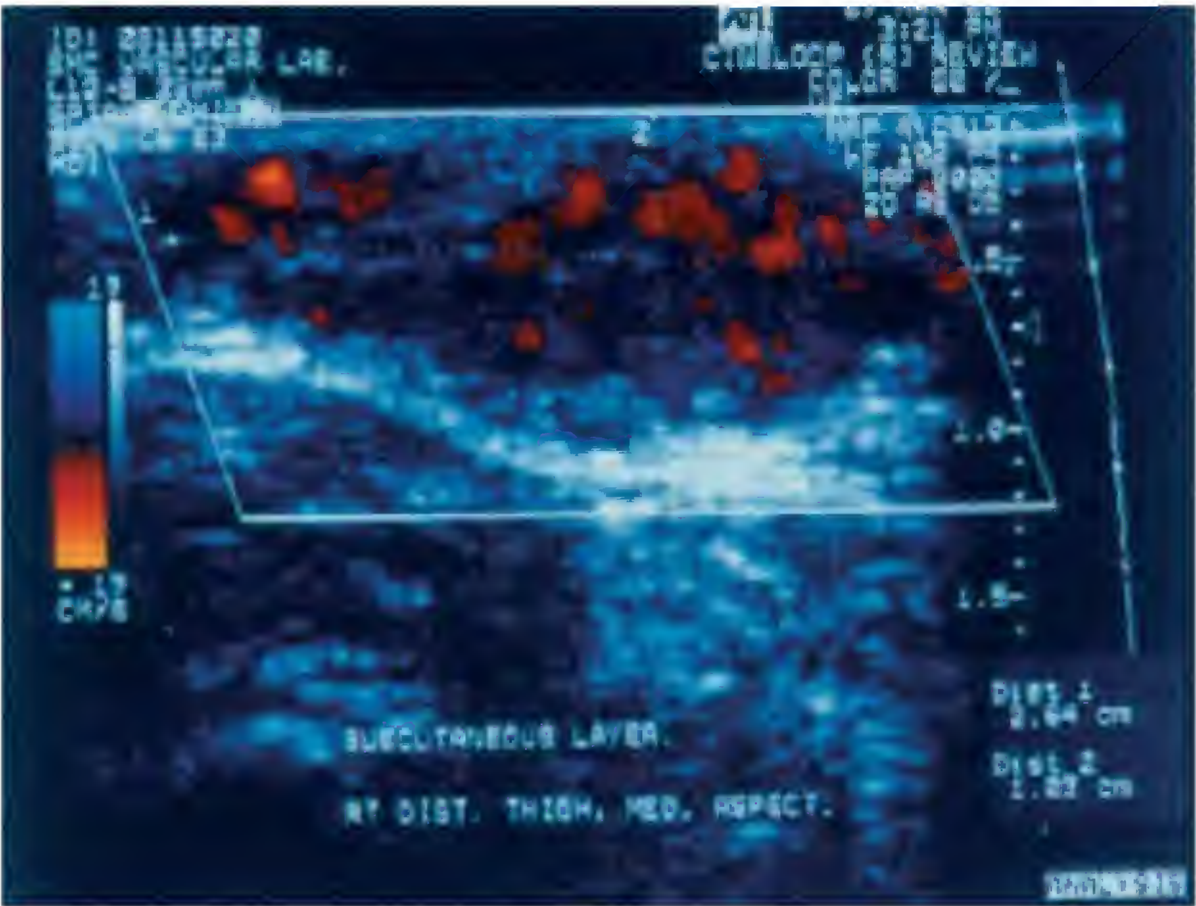
The VM in particular has a significant incidence of secondary abnormal long-bone growth with subsequent bone length discrepancy. In addition, it is also known to have a relatively high incidence of combined LM, which is still called Klippel–Trenaunay syndrome [15, 16].

Of the many clinical clues this patient presented with that suggested VM among various CVMs, immediate collapse of the bulging soft lesion along the foot upon elevation was the most important.

Therefore, hemodynamic assessment of the lower extremity along the scattered soft tissue tumors has to be the starting point for the work-up of proper diagnosis and treatment of this disease complex; duplex ultrasonographic study provides most of the essential hemodynamic information and an excellent guideline for further management (Fig. 41.2). **[Q2: C]**



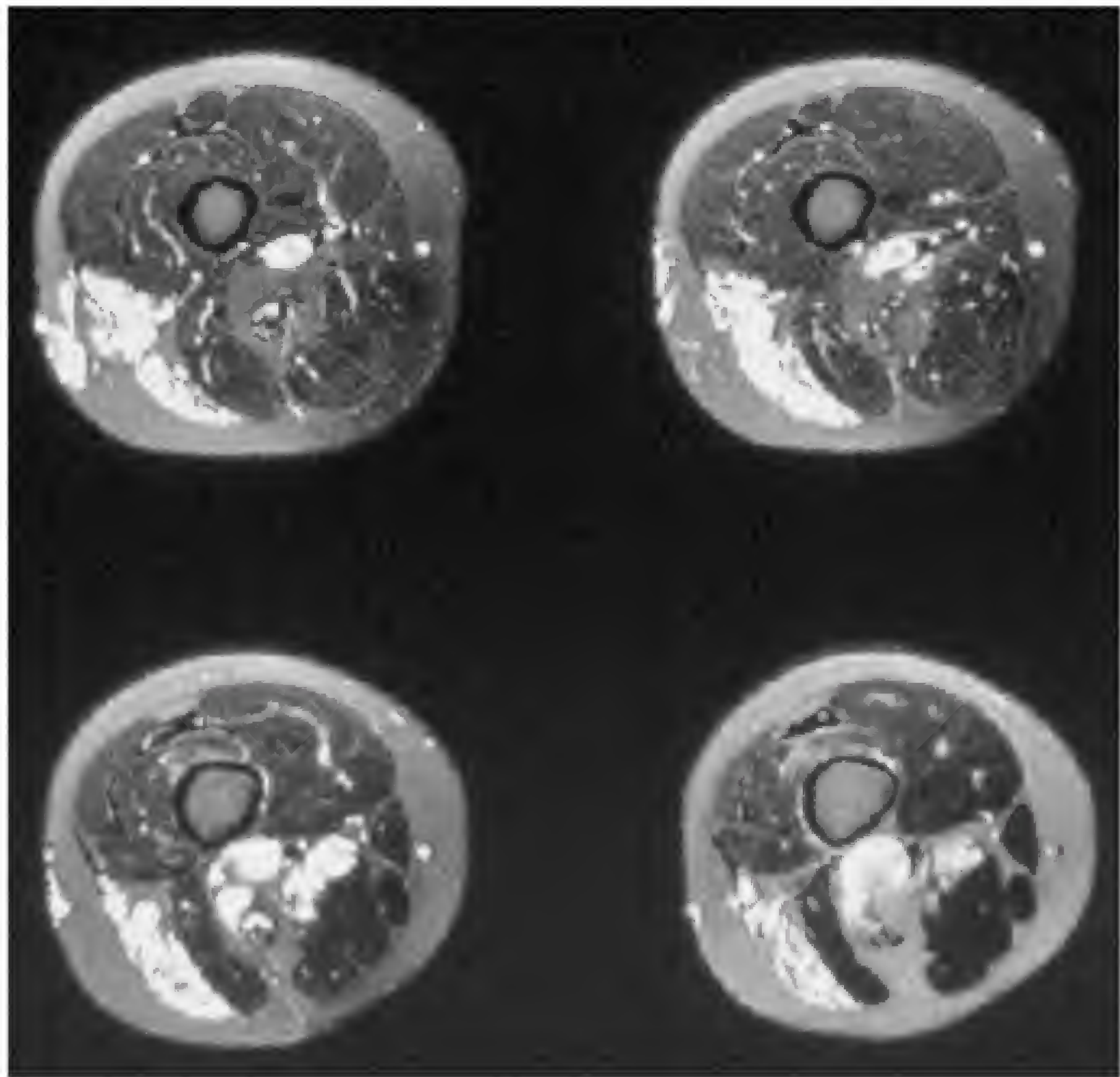
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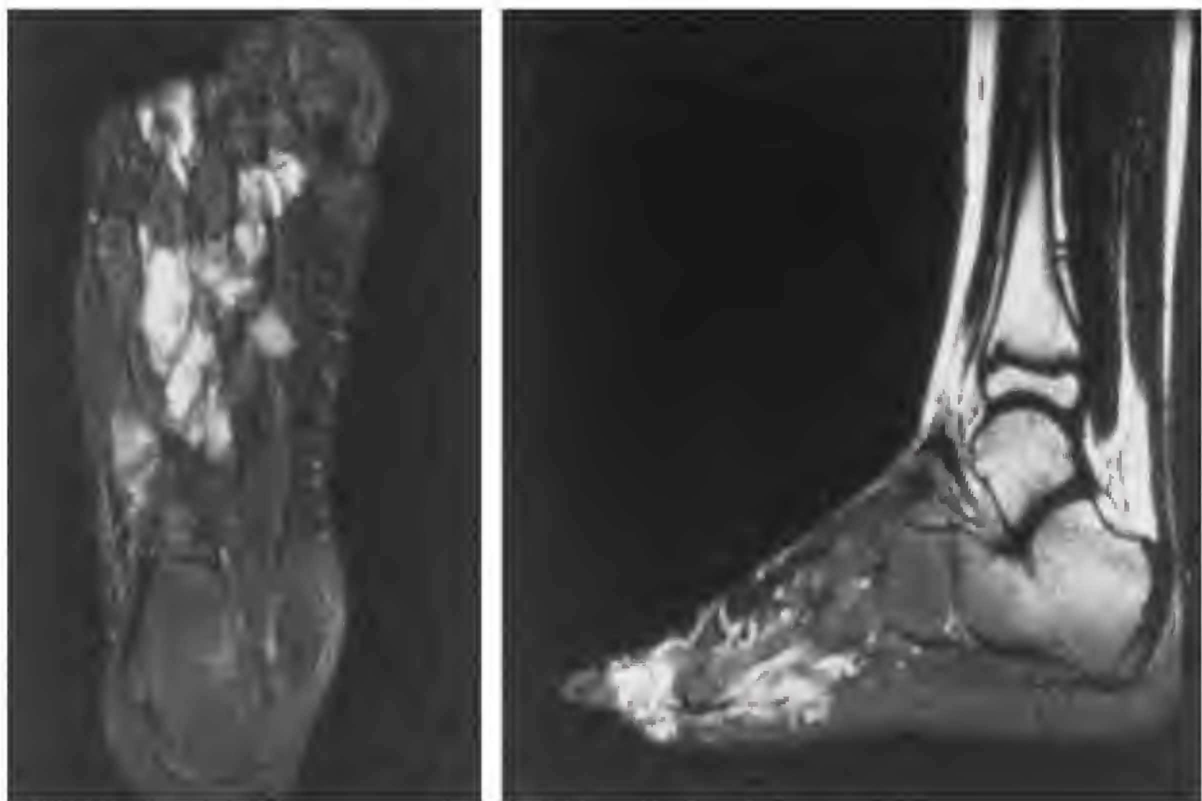
b

Fig. 41.2. a Sonographic identification of the communicating/draining vein between VM lesion and deep vein system. **b** Sonographic assessment of the VM lesion located superficially in the lower extremity.

Further study to assess scoliosis with pelvic tilt and/or abnormal long-bone growth with length discrepancy may be carried out once primary diagnosis of the vascular malformation has been made. In this case report, the patient presented



a



b

Fig. 41.3. a ET form of the VM in diffuse infiltrating status mostly confined within subcutaneous soft tissue, and T form of lesion along the deep vein system as femoral-popliteal venectasia. **b** ET form of the VM lesion, infiltrating into foot muscle structure as well as sole soft tissue.

recurrent episodes of tender swelling of the left knee following minor trauma. This was probably due to the bleeding/leaking from the VM lesion near to the knee joint to the surrounding soft tissue. A detailed evaluation of the knee joint itself can be deferred until the basic evaluation of VM, presented as soft tissue swelling along the knee joint, is completed with MRI, WBBPS, and duplex ultrasonography [17, 18]. This approach will delineate the accurate relationship of this VM lesion to the peri-articular structure including the joint space, and the potential risk of inducing hemarthrosis by repeated bleeding following trauma.

Radiological assessment of lumbosacral spine together with long-bone length discrepancy should be made *after* hemodynamic assessment to identify the extent of VM, starting with duplex scan as the most basic laboratory test [19].

Although duplex ultrasonographic study can provide most of the crucial first-line hemodynamic information about vascular malformation, MRI of T1 and T2 images is the most valuable non-invasive study for clinical diagnosis, and has become the new gold standard for the diagnosis, especially for the VM [17] (Fig. 41.3). **[Q3: C]**

MRI study of the soft tissue along the entire left lower extremity extending from toe to the torso can confirm the clinical diagnosis of VM already made preliminarily by ultrasonographic study. MRI can provide precise delineation of the anatomical relationship of the malformation lesion with its surrounding tissues like muscle, tendon, nerve, vessels, and bone from the foot to the retroperitoneal, pelvic, and gluteal regions. In addition to the duplex scan and MRI study in this patient, various non-invasive tests are needed for further differential diagnosis.

Lymphoscintigraphic study based on radioisotope-tagged sulfur colloid is indicated to assess lymphatic function and the lymph-conducting system in general and rule out chronic lymphedema due to the T form of LM [20, 21].

The extremity involved was felt to be firmer than usual for a VM-affected leg, with general diffuse swelling throughout the entire length of the lower limb; this finding suggested primary lymphedema combined with venous stasis so that further evaluation of the lymphatic function is indicated with radionuclide lymphoscintigraphy. The lymphatic function assessment of this patient with lymphoscintigraphy has shown the marginal status of the lymphatic system and its vulnerability to further insult by the ET form of LM.

WBBPS based on radioisotope-tagged red blood cell pooling is also indicated as one of three basic tests for the diagnosis of VM. This relatively new investigation is very sensitive in detecting abnormal blood pooling throughout the body (Fig. 41.4). It can be used not only as a practical test to assess treatment results but also as a screening test for hidden vascular malformation. It also has a unique role in the differentiation between venous and lymphatic malformation [22, 23].

CT scanning also has practical value in providing information on the relationship of vascular malformation to its surrounding skeletal and soft tissue of the lower extremity.

Transarterial lung perfusion scintigraphy (TLPS) can provide crucial information on possible involvement of a micro-, if not, macro-AVM lesion to the VM (Fig. 41.5).

AVM involvement is a critical condition for the management strategy of VM; the VLM in particular is seldom combined with the AVM, especially in micro-AVM, which can be overlooked by conventional arteriography alone. Positive confirmation of no existence of micro-AVM is extremely important before the initiation of the treatment to the symptomatic VM lesions, especially when it is combined with LM.

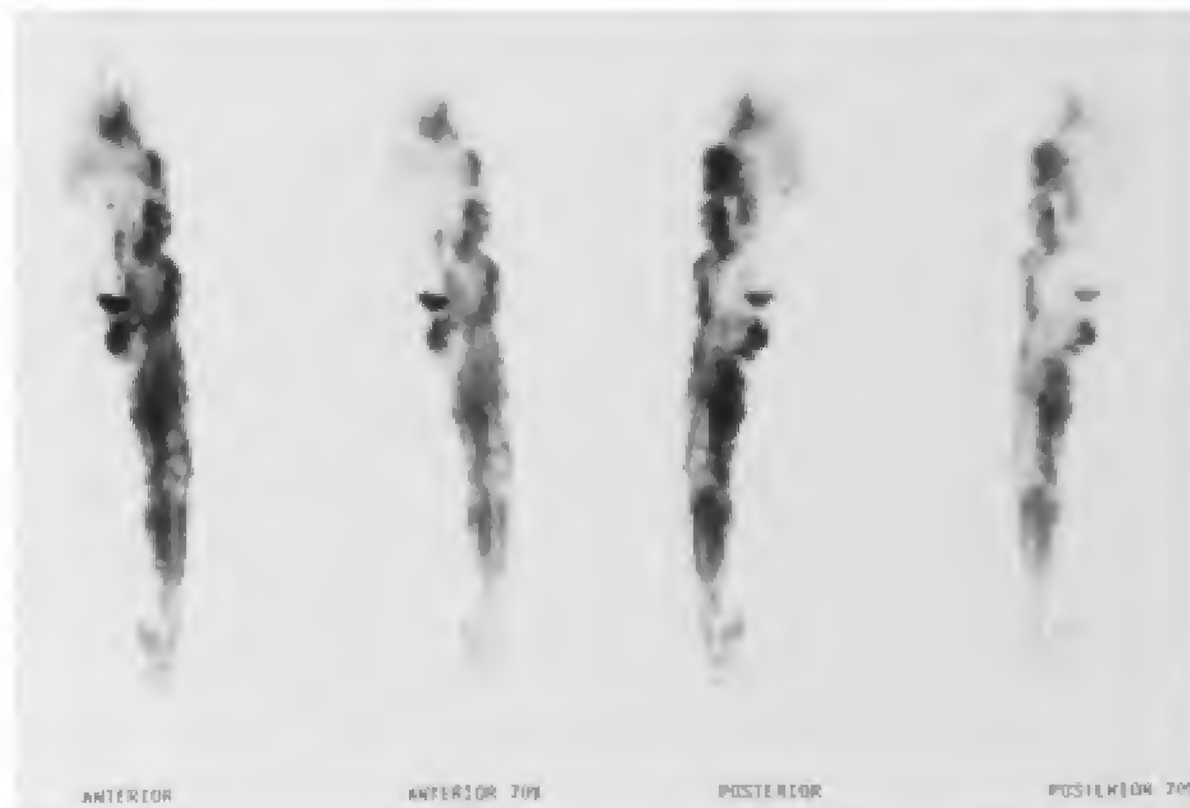


Fig. 41.4. Extensive abnormal blood pooling by the ET lesions and T lesion of the VM, diffusely involving entire lower extremity.

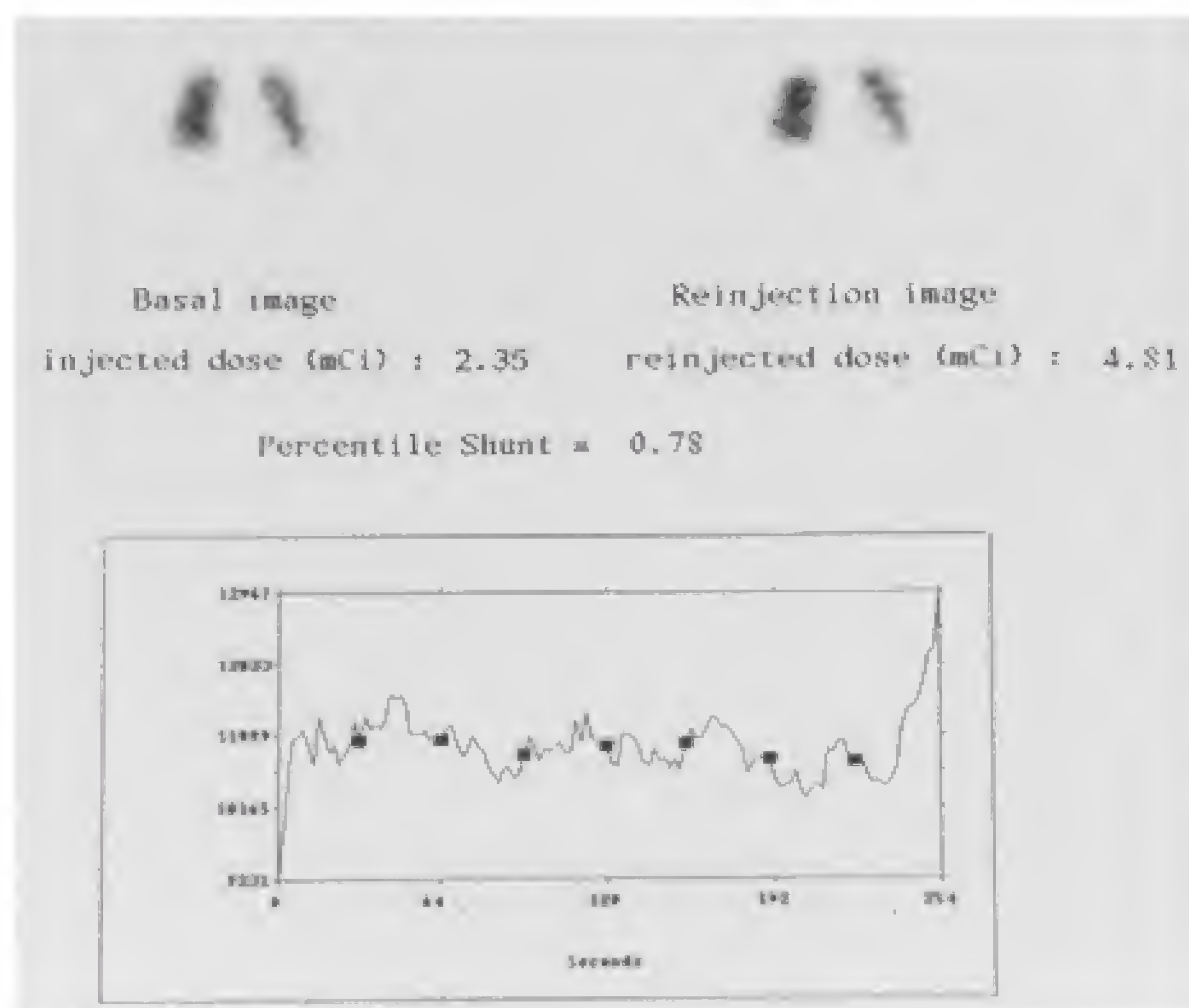


Fig. 41.5. TLPS investigation of arteriovenous (AV) shunting status in lower extremity to assess potential risk of the AVM lesion involved to the VM lesion. Normal TLPS finding with no evidence of micro-AV shunting can rule out AVM without further investigation by arteriography.

The TLPS can therefore provide necessary guidance for the further invasive study of arteriography [24, 25].

However, classical lymphangiography (or lymphography) using oil-based contrast material is *no longer* performed for the screening lymphatic function because of the potential risk of further damaging the lymphatic vessel with the procedure. **[Q4: E]**

Once the final diagnosis of a combination of VM and LM has been made, then the next decision should be whether treatment is indicated. In view of the abnormal long-bone growth involvement to this vascular malformation, immediate treatment of this particular VM is generally preferred.

Treatment priority should be given to the primary etiology, i.e. vascular malformation. Therefore, the control of abnormal hemodynamic status of the lower extremity secondary to the VM should have priority [26, 27]. **[Q5: C]**

All the other clinical problems secondary to this primary lesion, including scoliosis with pelvic tilt, abnormal long-bone growth with bone length discrepancy, and abnormal gait, can be deferred while treatment is aimed at the VM itself [5, 6, 26]. Not all the VM lesions are indicated or feasible for treatment. In general, VM lesions located near limb-threatening regions (e.g. proximity to the joint space) or potentially life/critical function-threatening regions (e.g. proximity to the airway), symptomatic lesions, and/or lesions with complications are generally considered for treatment [5, 11]. **[Q6: E]**

There is significant controversy over how to manage limb-length discrepancy as the secondary phenomenon of the VM in the lower extremity. Surgical intervention directly to the epiphyseal plate to arrest further abnormal growth of the affected long bone has brought mixed results, with further controversy on its long-term value [13, 14]. Therefore, general the consensus on this issue of vascular-bone syndrome accepted by most ISSVA members these days is to endorse a new strategy to control the hemodyamic abnormality of VM *first*, since hemodynamic impact/stimulation by the VM lesions to the intraosseous tissue along the epiphyseal plates is known to be the cause of abnormal long-bone growth [14, 26]. The strategy based on conservative treatment only with physical therapy and shoe adjustment until the long-bone growth is completed is also not acceptable due to increasing morbidity in gait and spine, as well as the unpredictable outcome of late correction. Meanwhile, too aggressive an approach with early correction of long-bone discrepancy has also been abandoned due to significant difficulty in achieving good long-term results. **[Q7: C]**

The traditional surgical approach of removing the entire lesion is still theoretically acceptable if the lesion is located in a surgically accessible area and localized enough to be completely removable with limited or no morbidity. However, this condition is generally very rare and for most VM lesions there will be significant morbidity with a surgical approach aimed at complete removal of the lesion.

Therefore, a multidisciplinary approach that combines traditional surgical therapy with newly introduced embolosclerotherapy utilizing various emboloscleroagents is the treatment strategy of choice [5, 6, 8]. This can substantially reduce overall treatment-related morbidity with good long-term therapy results [11, 12].

A lesion located along the surgically inaccessible area and/or with prohibitively high surgical morbidity is generally treated with sclerotherapy alone. The current trend in the management of VM of the lower extremity involves a multidisciplinary approach combining surgical therapy, sclerotherapy, and/or embolotherapy, whenever feasible [5, 27]. **[Q8: D]**

Most of the diagnosis of VM in the lower extremity in particular can be made efficiently on the basis of non-invasive studies. However, classical invasive studies, including arteriography and phlebography, are still considered to be the gold standard for the management of all vascular malformations, but they are generally reserved for use as a road map for the final therapeutic regimen (Fig. 41.6). These invasive imaging techniques are also used to rule out hidden micro-AVM combined with the VM, especially when TLPS findings indicate a high possibility of a micro-AV shunting condition [6, 8, 25]. **[Q9: C]**

Numerous emboloscleroagents have been tested for the treatment of VM; most recently, absolute ethanol has been accepted as the scleroagent of choice not only for VM but also for AVM, with excellent long-term outcome with no recurrence



Fig. 41.6 Percutaneous direct puncture phlebographic findings of the ET-form lesions of VM in the thigh; it may become a road map for the subsequent endovascular management with embolo/sclerotherapy.

when treated properly [11, 12, 25, 28–30]. However, this has significant side effects, resulting in various acute and/or chronic complications/morbidity, such as deep vein thrombosis, pulmonary embolism, nerve palsy, and various degrees of skin to soft tissue damage from bullae to full thickness necrosis. Therefore, the selection of ethanol as the scleroagent to treat VM has to be based on the risk involved of recurrence, acute morbidity during the therapy, and long-term sequelae of the treatment [6]. In order to treat VM of the lower extremity safely, careful hemodynamic assessment of the deep vein system is also mandatory, including confirmation of the existence of a normal deep vein system. This is crucial before treatment of the T-form lesion of VM, the marginal (lateral embryonic) vein in particular. Once the deep vein system is properly documented, proper treatment of VM can be initiated. **[Q10: D]** However, all the other issues raised in Question 10, including history of deep vein thrombosis, combined LM, and history of skin damage during previous sclerotherapy, will also require careful assessment to improve overall safety of the planned treatment.

Differential diagnosis with other forms, T or ET forms, as well as other kinds of vascular malformation, VM. LM, VLM, or AVM, is mandatory, in view of their different behavior with different clinical impact. This is particularly important for the ET form of various vascular malformations whose behavior is totally unpredictable. The ET form retains the original evolutional ability of mesenchymal cells, in contrast to the T form, so that it can grow when the condition/stimulation should meet (e.g. trauma, surgery, pregnancy, hormone therapy) [10]. Regarding the VM of the lower extremity, precise differential diagnosis of other conditions such as LM or AVM is extremely important, because the treatment strategy is substantially different [6, 27]. Besides, initial differential diagnosis for VM, like any vascular malfor-

mation, should start from the infantile (neonatal) hemangioma which also belongs to the vascular anomaly together with the vascular malformation. Hemangioma is a true vascular tumor and not a vascular malformation, possessing distinctively different pathophysiology, anatomico-histology, and clinical behavior [1, 31]. **[Q11: E]** The clinical significance of capillary malformation is not understood properly yet, but it should be included in the evaluation of any vascular malformation although the modified Hamburg classification did not include it in the classification of various CVMs, due to the lack of clinical significance for the vascular surgeon [32].

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42. Deep Venous Thrombosis

Fahad S. Alasfar, Dwayne Badgett and Anthony J. Comerota

A 67-year-old male had a history of a right calf deep venous thrombosis (DVT) following a flight from California to New York. He was treated on that occasion with anticoagulation with unfractionated heparin then Coumadin for 3 months. Recently, he was diagnosed with sigmoid cancer. He is now on postoperative day three from exploratory laparotomy, sigmoid colectomy and extensive lysis of adhesions. Although he was haemodynamically stable, he required a transfusion of three units of blood. DVT prophylaxis for the perioperative period included graded knee-high compressive stockings and intermittent pneumatic compression (IPC).

Question 1

What are the risk factors that predispose to DVT?

Question 2

What is the clinical presentation of a patient with anti-thrombin III (ATIII) deficiency?

Question 3

Regarding antiphospholipid antibody (APA) syndrome, which of the following is not correct?

A. Procainamide has been associated with the development of APA syndrome.

B. Thrombotic complications associated with APA syndrome are limited to the venous system.

C. Long-term anticoagulation has been recommended in managing APA syndrome, maintaining the international normalised ratio (INR) at 3 or higher.

D. Recurrent venous and arterial thrombosis is a major feature of the APA syndrome.

Question 4

Regarding Factor V Leiden gene mutation, which of the following is/are correct?

A. Factor V Leiden mutation is an important risk factor for pulmonary embolism and DVT during pregnancy or use of oral contraceptives.

B. Factor V Leiden mutation is associated with an increased risk of myocardial infarction and angina.

C. Hyperhomocystinaemia increases the risk of Factor V Leiden carriers having any Venous Thromboembolic Episodes (VTE) from two per cent to ten per cent.

D. A single-point mutation in the gene coding for coagulation Factor V results in the formation of a Factor V molecule that is not inactivated properly by activated protein C (APC).

Question 5

Which of the following statements are true concerning prophylaxis for DVT?

A. There are many prospective randomised studies supporting the efficacy of graded compression stockings in preventing DVT in patients with malignancy.

B. IPC is as effective as low-dose unfractionated heparin (LDUH) in reducing the risk of DVT.

C. LDUH and low-molecular-weight heparin (LMWH) are most effective in preventing DVT.

D. Dextran is an excellent alternative to LDUH in preventing DVT.

On the fifth postoperative day, the patient began complaining of mild left calf pain and swelling. On physical examination, his lower extremities were warm with normal pulses. The left calf was mildly swollen with slight tenderness. A venous duplex of the lower extremity revealed thrombosis of the left popliteal, posterior tibial and peroneal veins.

Question 6

Which of the following statements regarding perioperative DVT is/are correct?

A. In general surgery, the overall incidence of DVT as assessed by labelled fibrinogen uptake (FUT) is 25 per cent.

B. In surgical patients with malignant disease, the incidence of postoperative DVT is 60 per cent.

C. The incidence of postoperative DVT after total hip replacement is 45–55%.

D. Major trauma patients have a low risk for DVT.

E. Patients undergoing elective neurosurgical procedures have a 20–25% incidence of DVT documented by radioisotopic scanning.

The patient was started on a therapeutic regimen of LMWH (enoxaparin) 1 mg/kg every 12 h and a daily dose of Coumadin .The patient's baseline coagulation profile was normal and his platelet count was 190,000.On day three of anticoagulation, his INR was 2.2 and his platelet count dropped to 67,000.

Question 7

Regarding heparin-induced thrombocytopoenia (HIT), which of the following is/are correct?

A. It is caused by IgM antibodies that recognise the complex of heparin and platelet factor 4.

B. The peak incidence occurs 4–14 days after initiation of heparin.

C. It occurs more commonly with unfractionated heparin than with LMWH.

D. It can be treated by reducing the dose of LMWH.

E. Argatroban and hirudin are acceptable agents used for the treatment of HIT.

LMWH was discontinued and the patient started on Argatroban. On the tenth postoperative day, the patient started complaining of left flank pain and his haemoglobin level dropped to 6 g/dl.A computed tomography (CT) scan of his abdomen revealed a 6×7-cm retroperitoneal haematoma. Because of the haematoma, anticoagulation was discontinued and an inferior vena cava (IVC) filter inserted.

Question 8

Which of the following are acceptable indications for an IVC filter?

A. Complication or contraindication to anticoagulation in a patient diagnosed with a pulmonary embolism.

B. Recurrent thromboembolism despite therapeutic anticoagulation.

C. Acute iliofemoral DVT.

D. Recurrent pulmonary embolism with pulmonary hypertension.

Question 9

Regarding thrombolysis for acute DVT, which of the following is/are correct?

A. Studies show no difference in lysis capability between anticoagulation and lytic therapy.

B. Randomised studies support lytic therapy for all lower-extremity DVT.

C. Patients with iliofemoral DVT treated with catheter-directed thrombolysis have a better quality of life than patients treated with anticoagulation alone.

D. Lytic agents are more effective when delivered by catheter-directed intrathrombus infusion rather than systemic intravenous infusion.

Commentary

The natural history of DVT has been described well in the literature. Complications of venous thromboembolism continue to be a major cause of death and morbidity each year. In the USA, there are approximately 50,000–200,000 deaths each year secondary to pulmonary embolism. Fifty-two per cent of patients with DVT develop pulmonary embolism [1], most of which occur from the proximal venous segments of the lower extremities.

Patients with proximal DVT had a pulmonary embolism incidence of 66 per cent, whereas tibial thrombi had a 33 per cent incidence [1]. Multiple studies have shown a 50 per cent reduction in fatal pulmonary embolism when prophylaxis with LDUH is used [2]. Moreover, natural history studies have shown that the long-term morbidity of post-thrombotic syndrome (PTS) is significant following DVT. PTS has been reported in 33–79% of patients following proximal DVT and 2–29% of patients with calf DVT. Masuda et al. [3] reported valve reflux in 30 per cent of individuals with calf DVT followed for 3 years. Furthermore, they reported that 23 per cent of patients with calf DVT have ongoing pain and swelling of the affected extremity.

Thus, proper prophylaxis, early diagnosis and appropriate therapy are of paramount importance in preventing the short- and long-term complications of DVT.

An understanding of the risk factors for DVT is helpful for appropriate DVT prophylaxis. These risk factors include prior DVT/pulmonary embolism, prolonged immobilisation or paralysis, malignancy, major surgery (especially abdominal, hip and lower-extremity surgery), age over 40 years, and severe heart disease. There are also hypercoagulable states that predispose to thrombosis. Haematological abnormalities include protein C and protein S deficiency, Factor V mutation, disorders of plasminogen activation and antiphospholipid antibodies.

Lupus anticoagulant and HIT are also associated with DVT. Proteins C and S are part of the naturally occurring balance of coagulation that prevents thrombosis by inactivating Factors Va and VIIIa. Deficiency of these factors leads to an increased risk of thrombosis. Proteins C and S, like Factors II, VII, IX and X, depend on vitamin K. Because of the shorter half-life of protein C, a transient hypercoagulable state can be induced early in the course of treating patients with a warfarin compound due to the acute reduction in protein C level. A search for an underlying hypercoagulable disorder should be undertaken in patients with recurrent DVT or unexplained arterial or graft occlusion.

Chronic warfarin therapy may reduce the level of proteins C and S by 30–50%; therefore, these levels should be measured after the patient has discontinued warfarin. Indefinite oral anticoagulation is indicated in patients with confirmed deficiency. **[Q1]**

ATIII is an important naturally occurring anticoagulant that inhibits the enzymatic activation of thrombin and other naturally occurring clotting factors. The heterozygous form of ATIII deficiency is asymptomatic and may affect 1 in 2,000 people. A chronic deficiency of ATIII can occur with protein loss in nephrotic syndrome, liver disease, sepsis and Disseminated Intravascular Coagulation (DIC). When complications occur, heparin followed by Coumadin is the treatment of choice. **[Q2]**

APA is a heterogeneous group of circulating autoantibodies directed primarily against negatively charged phospholipid compounds. These antibodies interfere with the thromboplastin reaction against the activated platelet. Recurrent venous and arterial thrombosis is a major feature of APA syndrome. Thrombosis associated with APA syndrome has occurred in diverse anatomic locations, causing a wide spectrum of clinical manifestations. DVT and pulmonary embolism are common complications of APA [4]. Similarly, arterial thrombosis involving carotid [5], hepatic, splenic, mesenteric and retinal arteries causing infarction has occurred. APA syndrome should be suspected in young patients with stroke or arterial occlusion.

APA syndrome has been associated with multiple medications. However, procainamide has been implicated more commonly than other drugs [6].

The diagnosis should be suspected based on the clinical presentation or the unexplained prolonged PTT. Diagnostic tests for APA syndrome include serology testing for APA and clotting assays. The primary treatment remains anticoagulation, maintaining an INR at or above 3.0 [7, 8]. **[Q3: B]**

Protein C is one of the key regulatory proteins for coagulation cascade. APC cleaves and inactivates Factors Va and VIIIa. A single-point mutation in the gene coding for Factor V results in the formation of a Factor V molecule that is not inactivated properly by APC [9]. Factor V Leiden mutation is an important risk factor for pulmonary embolism and DVT, especially during pregnancy or oral contraceptive use [10].

Hyperhomocystinaemia increases the relative risk of a Factor V leiden carrier having any VTE [11]. There is no increased risk of myocardial infarction or angina in patients with Factor V Leiden mutation [12]. **[Q4: A, C, D]**

Among the available methods of DVT prophylaxis, LDUH and LMWH are the most effective in reducing DVT as assessed by FUT [13]. LDUH was the first antithrombotic agent evaluated in early randomised studies. LDUH, dextran, IPC and graded elastic stockings also significantly reduce the incidence of postoperative DVT [13].

LDUH given subcutaneously (5,000 U) every 8 or 12 h started preoperatively and continued postoperatively for 7 days has been shown to decrease the incidence of DVT from 25 to eight per cent [14]. Moreover, these studies have shown a 50 per cent reduction of fatal pulmonary embolism when patients are treated with LDUH. LMWH and LDUH have been shown to be equally effective in preventing DVT in general surgery patients [14].

Advantages of LMWH include improved bioavailability, once-daily dosing, and a lower incidence of HIT [15].

IPC is an attractive method of DVT prophylaxis since there are no observed complications. This device provides intermittent compression lasting 10 s/min with insufflation pressures of 35–40 mm Hg. In a trial comparing IPC with LDUH, both agents were effective in reducing lower-extremity DVT in high-risk patients [16].

Graded compression stockings decrease the risk of DVT, but data are limited regarding the effect on the prevention of DVT and pulmonary embolism. There are no randomised trials on the use of these stockings alone in high-risk patients, although current recommendations suggest the use of more effective methods. Fifteen to twenty per cent of patients will not receive benefit from elastic stockings because of their leg shape or size. Dextran has not been shown to be as effective as either LMWH or LDUH in preventing DVT; however, it may reduce the incidence of pulmonary embolism. Disadvantages of dextran include its high price, risk of anaphylaxis, potential for volume overload, and need for intravenous access. It is also contraindicated in patients with impaired renal and cardiac function. **[Q5: B, C]**

The incidence of DVT in general surgery patients has been well established. Overall, the incidence of DVT was 25 per cent in general surgery patients not receiving prophylaxis. In patients with other risk factors, i.e. malignancy, the risk of DVT is 29 per cent. Overall, the risk of pulmonary embolism is 1.6 per cent while the risk of fatal pulmonary embolism is 0.8 per cent [13].

Patients undergoing major orthopaedic surgery of the lower extremity are at high risk of postoperative DVT, despite improved techniques and early mobilisation. The incidence of postoperative DVT after total hip replacement is 45–57%, with the risk of proximal DVT being 23–36% [17]. The incidence of pulmonary embolism in this group is 6–30% and that of fatal pulmonary embolism is 3–6%. Because many pulmonary embolisms are asymptomatic, and because of the high incidence of DVT in the postoperative period, proper prophylaxis is mandatory [18].

DVT and pulmonary embolism are considered common complications after major trauma. A recent study using a venographic endpoint demonstrated that major trauma patients (injury severity score >9) have an exceptionally high risk of venous thromboembolism (58 per cent). This study also revealed that there is a greater than 50 per cent incidence of DVT in the major trauma subset [19].

Pulmonary embolism is the most frequent reason for death following spinal cord injury. Clinically recognised DVT and pulmonary embolism occur in only 15 and five per cent of cases, respectively [20]. However, the incidence of DVT in patients with acute spinal cord injury by venography has been reported to be between 18 and 100 per cent, with an average of 40 per cent. The incidence of fatal pulmonary embolism is 4.6 per cent, with the greatest risk occurring in the first 2–3 months after spinal-cord injury [21]. **[Q6: A, C, E]**

Approximately 2–5% of patients exposed to heparin will develop HIT. This is caused by IgG antibodies that recognise complexes of heparin and platelet factor 4, leading to platelet activation via platelet Fc gamma IIa receptors. Formation of a procoagulant, platelet-derived microparticles generates thrombin and makes patients especially vulnerable to venous thromboembolism [22, 23].

When examined directly, the clot appears white due to the concentration of fibrin and platelets. HIT should be suspected if a patient develops DVT or pulmonary embolism while receiving heparin, especially if the platelet count drops below 35 per cent. HIT usually develops between the fourth and fourteenth days after initiation of heparin, although a rapid fall in platelet count can occur in response to heparin if the patient has had recent heparin exposure.

HIT occurs much more commonly with unfractionated heparin than with LMWH [15]. Upon recognition of HIT, heparin should be discontinued; however, appropriate anticoagulation should be continued to avoid a thrombotic complication, which

has been observed in up to 50 per cent of patients within 30 days of the diagnosis of HIT [15].

Current treatment options include lepirudin [24], argatroban and danaparoid. Lepirudin is recombinant hirudin (r-hirudin) and is approved for the treatment of patients with HIT. It is a potent direct thrombin inhibitor and is given in a bolus dose of 0.4 mg/kg/min followed by an infusion of 0.2 mg/kg/h, but the dosage should be adjusted in patient with renal dysfunction. Argatroban is a synthetic peptide that binds to and inhibits thrombin. It is given in doses of 0.5–4 μg/kg/min and has the advantage of normal excretion (hepatic) in patients with impaired renal function. Danaparoid is a mixture of heparan sulphate and dermatan sulphate, which inhibits thrombin generation indirectly via inhibition of Factor Xa, with some direct anti-thrombin activity as well. The disadvantage of danaparoid includes a 10–20% in vitro cross-reactivity with HIT antibodies and long half-life. **[Q7: B, C, E]**

IVC filters are intended to prevent pulmonary emboli following filter insertion. Anticoagulation should be continued whenever possible to prevent further thrombosis [25, 26]. The primary indication for the insertion of an IVC filter is the occurrence of a complication of or contraindication for anticoagulation therapy. Less frequent indications for the insertion of an IVC filter are recurrent thromboembolism despite adequate anticoagulation therapy and chronic recurrent pulmonary embolism with pulmonary hypertension.

Finally, IVC filters have been used for pulmonary embolism prophylaxis in patients with proximal DVT who are at high risk for bleeding and selected trauma patients (pelvic fracture) who are at high risk for VTE and cannot be managed with effective prophylaxis. **[Q8: A, B, D]**

Restoring patency by eliminating the thrombus in the deep venous system is the ideal goal of therapy for acute DVT. Many reports have shown that lysis can be achieved and patency restored with thrombolysis, and that long-term sequelae occur less often with successful treatment. Systemic thrombolytic therapy for lower-extremity DVT is associated with a 40–60% success rate. While recanalisation is better than standard anticoagulation, the increased risk of bleeding complications has reduced enthusiasm for thrombolysis.

It has been shown that patients with iliofemoral DVT treated with catheter-directed thrombolysis have better functioning and wellbeing than patients treated with anticoagulation alone [28]. Currently, it is recommended that lytic agents be delivered via catheter-directed technique into the thrombus. Thrombolytic therapy is recommended for patients with iliofemoral DVT and selected patients with infrainguinal DVT who are severely symptomatic due to multilevel thrombosis. **[Q9: C, D]**

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43. Primary Varicose Veins

Michael Dialynas and Stephen G. E. Barker

A 39-year-old female sales assistant presented with a lengthy history of a dull ache affecting her right leg. She complained also of occasional night cramps and mild ankle swelling. Usually, the ache began mid-morning, became progressively worse during the course of the day and was exacerbated further with prolonged standing and in warmer weather. She had noticed also that the symptoms became worse following strenuous exercise. The patient attributed these symptoms to her rather unsightly, dilated, tortuous veins present throughout the leg. Otherwise, her medical history was unremarkable. She was not on any regular medication.

Question 1

At this stage, what else would you like to know from the history?

A. How long has she been aware of her "varicose" veins?

B. Are there any other members of the family with varicose veins?

C. Is there any history of superficial, or deep venous thrombosis?

D. Is she taking an oral contraceptives?

The patient had first noticed her varicose veins in her late twenties, but she was certain that they had become far more prominent following her second pregnancy. During this pregnancy, she was told that she may have had "a clot in the right leg", but she could not remember the exact diagnosis or whether she had had any treatment for it. On current inspection, there were prominent varicose veins affecting her right leg, mainly below the knee, on the anteromedial aspect, but with varices sited posteriorly, in addition. Multiple cutaneous telangiectases were present. The left leg appeared quite normal. Both her mother and eldest sister suffered from varicose veins.

Question 2

Identify which of the following statements are true or false.

A. Premenopausal women have a higher prevalence of varicose veins.

B. Age, gender, parity, race and occupation are factors contributing to the development of varicose veins.

C. Leg oedema is a prominent feature of varicose veins.

D. In the presence of a varicose long saphenous vein (LSV), the saphenofemoral junction is always incompetent.

E. Cutaneous telangiectases are only ever associated with varicose veins.

F. Patients with varicose veins are more likely to complain about unsightliness, rather than any other specific symptom.

Question 3

At this stage how would you assess the patient?

A. Examine clinically using a tourniquet test.

B. Examine clinically using a hand-held Doppler.

C. Request a venogram.

D. Measure the ankle brachial pressure index (ABPI).

E. Request a venous duplex scan of both legs.

On examination, with the patient lying flat and having raised the limb to drain the blood within the veins, a tourniquet was placed around the upper thigh (the Trendelenburg test). On standing the patient, varices below the applied tourniquet filled very quickly. No further distension was noticed, however, when the tourniquet was released. A second application of the tourniquet, just above the knee, controlled those varices sited anteromedially on the leg, but did not control those sited more posteriorly. Examination using a hand-held, 8-mHz, directional Doppler system suggested reflux in the lower LSV and in the short saphenous vein (SSV), behind the knee. However, the Doppler findings were not suggestive of reflux at the saphenofemoral junction, in the groin.

Later, a formal venous duplex ultrasound scan was requested. This confirmed incompetence and reflux within the LSV, but in the presence of a competent saphenofemoral junction. An incompetent perforating vein was demonstrated just above the knee (a Hunterian perforator) and its position was recorded. Other perforating veins lower down the leg appeared normal, with no evidence of reflux. Varices associated with the SSV were identified, together with an incompetent saphenopopliteal junction, sited some 5 cm above the popliteal skin crease. The deep venous system was patent and competent, with no signs of other abnormality. Hence, the varicose veins present were classified as “primary”.

Question 4

Identify which of the following statements are true or false.

A. The Trendelenburg test is a reliable method for assessing valvular incompetence and reflux.

B. Incompetent perforating veins are detected accurately with clinical examination alone.

C. Duplex ultrasound scanning constitutes the "gold standard" for investigating venous anatomy, incompetence and reflux.

D. Descending phlebography is a more accurate method of assessing the deep venous system than duplex ultrasound scanning.

E. The Hunterian perforating vein is the only common "communicating" vein in the lower limb.

Qustion 5

How would you treat this patient?

A. There is no clear indication to offer any treatment.

B. Offer injection sclerotherapy.

C. Offer below-knee compression stockings.

D. Offer surgery.

The patient was advised that the "disease" progression is very slow. However, the varicosities are likely to "deteriorate" with age and therefore it is likely that some active measures would need to be taken. Patient compliance and some change in lifestyle, along with conservative measures such as compression stockings, seem to provide symptomatic relief in the majority of cases (often reducing the need for surgery).

The patient opted for surgical treatment, under general anaesthesia. On admission and after obtaining patient consent, the operating surgeon marked the position of the varicose veins, with an indelible ink pen, with her standing. The previous duplex ultrasound scan report was reviewed, with attention paid in particular to the position of the incompetent perforating vein and the saphenopopliteal junction.

An appropriate consent was obtained from the patient. On transfer to the operating theatre, the patient was placed prone. The skin was prepared with a non-alcoholic, iodine-based solution. Sterile drapes were placed as appropriate first, for exploration of the saphenopopliteal junction. A transverse incision was made above the popliteal skin crease, where the saphenopopliteal junction had been identified previously by the duplex ultrasound scan. The saphenopopliteal junction was identified formally, with the artery adjacent. The SSV was ligated and divided almost flush to the junction. A short segment of SSV was excised (although the SSV was not formally stripped). Multiple avulsions of the previously marked varicosities were performed (using an Oesch hook), through 1–2-mm skin incisions. After

closure of wounds, the patient was placed supine, with the legs abducted and with head-down tilt (Trendelenburg position), to help reduce any intraoperative blood loss. The skin was re-prepared and new drapes were placed appropriately. A short, oblique incision was made in the groin crease, near to the saphenofemoral junction, and by careful dissection, the saphenofemoral junction was identified formally. Subsequently, all tributaries to the LSV were ligated (using absorbable sutures) and divided and in particular, the deep pudendal vein(s). Formal exposure of a small length of the femoral vein enabled ligation of a small tributary. Following this, the saphenofemoral junction was ligated almost flush to the femoral vein and the LSV divided. A disposable vein stripper was inserted intraluminally and advanced down the LSV, to just below the knee. A small skin incision (in Langer's lines) was made down onto the palpable tip of the stripper, to exit it. The LSV was stripped downwards. Varicose veins lower in the leg were avulsed via multiple, 1–2-mm skin incisions, using an Oesch hook. The Hunterian perforator noted and marked previously was ligated individually and subfascially through a small incision placed directly over it. The groin wound was closed (inclusive of a subcuticular suture) and Steri-Strips alone were applied to all the small stab incisions. A local anaesthetic agent was injected in the wounds to facilitate postoperative pain control. To finish and with all wounds closed, the whole leg was bandaged firmly, including the foot. Later, the bandage was exchanged for a full-length, class II graduated compression stocking that the patient was advised to wear for 6 weeks. The patient was reviewed 6 weeks postoperatively. All the wounds had healed and there were no residual varicosities.

The patient re-presented 5 years following the initial surgery with recurrent varicose veins in the same leg. She described symptoms similar to what she had prior to the initial operation, though to a lesser degree. The patient was concerned and requested treatment.

Question 6

How would you assess the patient?

A. Examine clinically using a tourniquet test.

B. Examine clinically using a hand-held Doppler.

C. Request a venogram.

D. Request photoplethysmography (PPG).

E. Request a duplex scan.

The patient was clinically examined using the tourniquet test and also a hand-held Doppler. A long varicose vein in the anteromedial aspect of the thigh and also a few varicose veins in the anteromedial aspect of the lower leg were noted. Some reflux was evident in the groin but not in the popliteal fossa when examined with the hand-held Doppler.

A duplex scan was requested to assess the anatomy of the varicosities further. This demonstrated a small anterior, incompetent, thigh vein originating from the groin (communicating with the femoral vein) that was feeding the lower varicosities. No perforators or saphenopopliteal incompetence were noted.

Question 7

How would you treat the patient given these findings?

A. Offer conservative treatment with compression hosiery.

B. Proceed to surgery.

C. Review in 6 months.

D. Offer sclerotherapy.

The options of treatment were again discussed with the patient as in the initial consultation. It was explained that surgery is thought to provide a more definitive treatment than other methods, albeit with an increased risk of complications for recurrence. The patient was not keen on symptomatic control with compression stockings. She wished to have more definitive treatment than that with sclerotherapy or local ablation of the visible varicosities and opted for surgery under general anaesthesia.

The patient was appropriately consented and informed about the increased risks of recurrent groin dissection, in particular the risk of damaging the femoral vein. Prior to the operation the recurrent groin vein was marked over the skin of the patient with a permanent marker pen, using duplex scan, to facilitate dissection.

The patient was then placed in the operating theatre, the skin prepared and draped as described previously for the initial operation. An incision was made over the previous scar and careful groin dissection was undertaken starting more medially where there was more virgin territory towards the previously marked vein. The recurrent anterior thigh vein was identified and ligated flush with the carefully exposed femoral vein. No further tributaries were noted. The recurrent vein was then stripped to as low as possible as described before. Multiple avulsions of the rest of the varicosities were also undertaken to complete the procedure. The leg was then bandaged as described in the initial operation. The bandages were later exchanged to a full-length class II graduated compression stocking that the patient was advised to wear for 6 weeks.

Commentary

This case is representative of the very many patients seen in varicose vein clinics. Patients with varicose veins are very likely to complain about unsightliness, but in addition, may focus on symptoms such as aching (less commonly pain), night cramps, “itching”, mild ankle swelling and, perhaps, bleeding from varicosities (usually following trauma). **[Q2: F = true]** Symptoms often become worse towards the end of the day and in warmer weather. In women, varicose veins may become more prominent during menstruation and commonly during pregnancy [1, 2].

Varicose veins associated with the LSV present usually, in the anteromedial and posterior aspects of the thigh and in the suprapatellar region. In the lower leg, LSV-associated varicosities are commonly seen antero- and posteromedially. SSV varicosities are most commonly situated along the posterolateral aspect of the lower leg. The presence of significant lower limb ankle oedema is not a common feature of varicose veins and more general causes such as cardiac failure, nephrotic syndrome, or other fluid retention syndromes should be considered [1, 2]. **[Q2: C= false]**

Superficial thrombophlebitis of varicose veins is not uncommon. The patient may present with tenderness along the line of any inflamed superficial vein. Rarely, superficial thrombophlebitis may extend to the femoral vein, resulting in a deep venous thrombosis (DVT).

Cutaneous telangiectases (dilated, intradermal venules) may be present. They are more common in women and often develop during pregnancy or at the menopause. They may extend to cover extensive regions of the leg, causing blue-purple discoloration of the skin. They are not true varicose veins, but represent a separate and distinct entity which may, or may not, be directly associated with varicose veins. Paradoxically, they can appear more profoundly following varicose vein surgery [1, 2]. **[Q2: E = false]**

Prolonged "venous hypertension" within the varicosities can cause skin changes later on, mainly around the lower third of the leg (the "gaiter area"), which can range from mild erythematous pigmentation (with haemosedirin deposition), to full-blown lipodermatosclerosis (although this is more commonly associated with a post-phlebitic limb), or even ulceration. Occasionally, lipodermatosclerosis may appear in an acute form as a painful, thickened and sometimes raised tender area over the lower leg, with no associated pyrexia, or lymphadenopathy. It can resolve spontaneously, or progress to a more chronic form [3].

A number of epidemiological studies from both Europe and North America have shown the prevalence rate of varicose veins within the general population to be approximately 2 per cent. Women seem to be affected more than men. **[Q2: A = true]** However, the incidence of varicose veins seems to increase with age, with a peak between 50 and 60 years [4]. Being multiparous, having an occupation involving prolonged standing and being of excessive height and weight are all thought to contribute to the development of varicose veins. **[Q2: B = true]** Post-thrombotic damage to deep veins, pelvic tumours and arteriovenous fistulas are all implicated in the development of (secondary) varicose veins. All patients presenting with primary varicose veins should have a careful history taken and be examined to exclude the (rare) presence of congenital syndromes (such as Klippel–Trenaunay and Parkes–Weber syndromes) and more importantly, any history of previous DVT. Even though the genetic predisposition for varicose veins seems uncertain, there does appear to be a strong association within families. **[Q1: A, B, C]**

Primary varicose veins are not always associated with an incompetent saphenofemoral junction. A recent survey of patients attending a major teaching hospital in London demonstrated (on duplex ultrasound scanning) that 42 per cent of cases presenting with primary varicose veins had in fact an intact saphenofemoral junction [5]. Varicosities can appear in isolation, or might be fed from incompetent perforating veins (including Hunterian and Boyd's perforators and those found at various intervals above the medial malleolus), in addition to those found because of saphenofemoral incompetence. **[Q4: E = false]**

The distribution of varicosities down the leg is unlikely to define with certainty their origin from either the LSV, or SSV. When examining patients, the tourniquet test (Trendelenburg test), being operator dependent, can not always provide accurate information as to the presence and location of incompetent perforating veins [6]. **[Q4: A, B = false]** The use of a hand-held, directional Doppler ultrasound system is a quick and easy way to assess incompetence of the LSV when clinical signs are equivocal, but it is not reliable when assessing for perforating veins, or the SSV. There are a number of investigations available to assess venous insufficiency further. The use of invasive diagnostic tests such as phlebography, to assess incom-

petence of perforating veins and the deep venous system, even though commonly used in the past, is probably not justified nowadays. Although phlebography is considered specific, its specificity is poor when compared to duplex ultrasound scanning [7]. **[Q4: D = false]** The optimum, non-invasive method to investigate venous anatomy, reflux and incompetence is duplex ultrasound scanning. Its value in also assessing recurrent varicose veins, short saphenous incompetence and perforating vein incompetence is well established and its use should be regarded as the new "gold standard" for venous examination [8]. **[Q4: C = true] [Q3: A, B, D]**

When treating varicose veins, the clinical objectives should be satisfactory cosmesis, relief of symptoms and prevention of complications and recurrence. Treatments should be discussed on an individual basis and may be non-surgical or surgical. Simple reassurance may be all that is required for some patients. For others, application of fitted, elasticated, graduated compression stockings may provide enough relief to avoid surgical or other intervention. Injection sclerotherapy aims to obliterate varicose veins by placing an irritant solution directly within the vein lumen, causing a local chemical reaction, promoting thrombosis. There are a number of commercially available sclerosing agents, with more or less similar properties. Many advocates for the method use it to treat all degrees of varicosity, with the "French school" injecting sclerosant directly into the uppermost portion of the LSV to obliterate its termination. However, the results are highly variable and operator-dependent and there is always the risk of DVT if the agent disperses into the deep venous system. Extravasation of the sclerosant can cause local irritation and pain and on occasion, pigmentation and ulceration of the overlying skin. Recurrences with this method of treatment are frequent [9, 10]. **[Q5: B, C, D]**

Surgical treatment for long and short saphenous varicose veins is performed (usually) under general anaesthesia. The procedure can be undertaken on a day case basis for those patients who are suitable (often only single leg). Various techniques such as ligating the saphenofemoral junction only, obliterating the LSV near to the junction using either chemical agents, or even electrical current, have been used as a method of treatment with varying success. The most acceptable surgical procedure, however, is near-flush ligation and division of the saphenofemoral junction, ligation of all the tributaries and stripping of the LSV to just below the knee. The procedure is completed with ligation (or avulsion) of incompetent perforating veins and avulsion of varicosities via stab incisions [11].

Preoperative identification and positional marking of incompetent perforating veins, with duplex ultrasound, is an imperative step to help prevent recurrence and facilitates the ligation, avoiding unnecessarily long scars [12].

SSV reflux and saphenopopliteal incompetence is treated using similar principles. Near-flush ligation of the saphenopopliteal junction is important. However, as the anatomical position of the junction can vary considerably, preoperative marking using duplex ultrasound scanning is essential. The junction may be "behind the knee", in the skin crease, but often, it can be many centimetres higher. Definitive exposure of the main popliteal vein, adjacent to the artery, should be attempted. Stripping of the SSV is considered unnecessary by many surgeons, but excision of a 2–5-cm length of vein helps prevent the possibility of recurrence.

Significant postoperative complications following primary varicose vein surgery include: haematomas from slipped ligatures, unligated vessels, injury to the femoral vein, injury to the femoral artery and development of a lymphocele. During exploration of the saphenopopliteal junction, injury to the popliteal vein and artery can occur. Furthermore, damage to the sural, the saphenous and lateral popliteal

nerves can occur while stripping veins and avulsing varicosities. The development of a DVT is a potential, but fortunately rare, complication of varicose vein surgery. More general surgical complications such as wound infection and dehiscence can occur also. Damage to major vessels should be repaired by direct suturing, patching, or even grafting where necessary. Lymphoceles, in general, regress with time. Sural or more commonly saphenous nerve damage was a frequent complication when the LSV used to be stripped upwards from the ankle. However, stripping downwards to just below the knee is safer, as the nerve is quite distant from the vein at this level. Care should be taken when avulsing varicosities as it is possible to damage adjacent nerves [13].

Recurrence of varicose veins constitutes approximately 20 per cent of the total operations undertaken for varicose veins overall. Various reports have demonstrated recurrence rates of anywhere between 5 and 80 per cent, within 5 years [14, 15]. Failure to ligate and divide the LSV and all its tributaries has been shown to be associated with the highest recurrence rates. Recurrences occur also when incompetent perforating veins go unrecognised. An alternative mechanism to explain recurrence, "neovascularisation", was suggested by Glass; new veins could develop through growth of newly formed vessels, or through widening of small, pre-existing collaterals. Re-canalisation of ligated, but undivided, vessels has also been suggested as a possible mechanism. Clinical examination follows the same principles as for primary varicose veins. **[Q6: A, B, E]** The management of recurrent varicose veins follows the same pathway as with primary varicose veins, with surgery being the most definitive treatment. Many patients seek simply reassurance about the potential risks of ulceration and deep venous thrombosis and decide to have only symptomatic treatment with graduated compression stockings.

The indications for sclerotherapy for primary and recurrent varicose veins depend upon the size, site and extent of the varicose veins, the presence of reflux points as well as the functional state of the main vein trunks. If the main vein trunks are incompetent, sclerotherapy on its own is unlikely to provide a long-term satisfactory result. Here, it should be considered as a useful adjuvant therapy to surgery, but not a complete treatment on its own. **[Q7: A, B, D]**

With recurrent varicose veins, a preoperative assessment using duplex ultrasound scanning is essential. Surgical treatment, if indicated, should be performed by an experienced vascular surgeon, as injury to the major vessels in the groin is far more likely [16, 17].

The most effective and long-lasting treatment for primary and recurrent varicose veins remains surgery, even though it can become a compromise between the attempt to remove the diseased veins and the need for an acceptable cosmetic result. It is imperative that the operation is performed properly in the first attempt by an appropriately qualified surgeon following all the principles outlined previously. Further research into the causes of recurrence may provide information on how to manage the condition more effectively.

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44. Venous Ulcers Associated with Deep Venous Insufficiency

Seshadri Raju

A 46-year-old female schoolteacher and non-smoker presented with an ulcer on the medial side of the ankle. The ulcer had persisted for the past year despite compressive dressings at a hospital wound care center. Ulcers in the same general area had occurred intermittently in the past but had healed with local wound care and dressings. The ulcer was very painful, particularly with dependency of the leg (7/10 over a visual analogue scale) and frequently at night. The patient had made a habit of elevating her legs during the day whenever feasible, and to sleep with her legs elevated on a pillow at night. She had been using a non-steroidal anti-inflammatory drug once or twice a day at work for pain relief, but lately a narcotic prescribed by her physician was required for sleep at night. Even so, on some nights, she had to "walk off" the pain for twenty to thirty minutes before she could fall asleep.

Past medical history: She had been hospitalized on two occasions during the past year for cellulitis of the leg, which required intravenous antibiotics. Her saphenous vein was stripped 15 years ago when the ulcer initially appeared. This resulted in healing of the ulcer but it recurred 2 years later. During adolescence, she sustained a closed tibial fracture of the same extremity during a ski accident and was in a plaster cast and crutches for several weeks.

Family history: No one in the family had varicose veins or deep venous thrombosis.

Examination: The patient was found to be healthy except for the affected extremity, which had a large 5 × 10-cm indolent ulcer on the medial aspect of the lower third of the leg. The ulcer bed had clean granulation tissue with serous drainage. The ulcer was surrounded by a broader area of hyperpigmentation in the "gaiter" area. No obvious varicosities or "blow outs" were noted. Good pedal pulses were present.

Question 1

Which of the following is *least* likely in this patient?

A. "Primary" deep vein valve reflux.

B. Post-thrombotic syndrome.

C. Popliteal artery entrapment.

D. Recurrent saphenous reflux from neovascularization.

E. Perforator incompetence.

The patient was referred to the vascular laboratory, where a detailed duplex venous examination was performed. Extensive reflux throughout the deep venous system in the affected extremity was found. Both the femoral and popliteal valves were refluxive, with valve closure times of 7 s and 6 s, respectively. The great saphenous was confirmed absent with no evidence of tributary or collateral reflux around the short sapheno-femoral stump. Neovascularization was not detectable. No significant perforator reflux was found, and the short saphenous vein was not refluxive. The deep venous system was widely patent without evidence of prior thromboses.

Air-plethysmography (APG) results were as follows: venous filling index (VFI_{90}) 7 ml/s; venous volume (VV) 135 ml; ejection fraction (EF) 60 percent; residual volume fraction (RVF) 48 percent.

Based on the above findings and the clear failure of conservative therapy to heal the ulcer, surgical intervention was discussed with the patient. She consented to this approach. Other preoperative work-up included a hypercoagulation profile and ascending and descending venography.

Question 2

Which of the following statements is true?

A. Duplex is more specific than descending venography in assessing reflux.

B. Valve closure time (VCT) is a reliable quantitative measure of reflux.

C. Venous filling index (VFI_{90}) with APG correlates best with ambulatory venous pressure.

D. Absence of varicosities or “blow outs” on physical examination rules out neovascularization or perforator reflux as a significant source of reflux.

E. Palpable pedal pulses rule out arterial insufficiency as the etiology in patients with painful leg ulcer.

The patient underwent internal valvuloplasty (Kistner technique) of the femoral vein valve under general anesthesia. Postoperative recovery was uneventful. DVT prophylaxis included low-molecular-weight heparin (LMWH) started preoperatively and continued until discharge, intraoperative intravenous heparin (5000 units), and daily warfarin sodium. Pneumatic compression was started during surgery and continued postoperatively when not ambulatory. She was discharged on 5 mg warfarin with instructions to the local physician to maintain the international normalized ratio (INR) at or above 2.5 for 6 weeks, after which the dosage could be lowered for a target INR of 1.7–2.0. The patient was instructed to wear elastic stockings for at least 6 weeks on a daily basis, after which she could adjust the usage as desired.

The patient was seen on follow-up at 6 weeks, at which time the surgical incision was well healed and the ulcer had become epithelialized to 90 percent of the original surface area. She requested and was granted permission to go back to full-time work. When seen in follow-up at 4 months, the patient reported that the ulcer had healed completely 2 weeks after the first clinic visit and had remained healed since. She was free of pain and had abandoned regular use of her stockings. She found it necessary to use them only occasionally when she expected her day to be more strenuous than usual. Physical examination revealed good-quality skin coverage over the previous ulcer, and the limb was free of edema. Interval follow-up duplex examination showed competence of the repaired femoral valve with valve closure time of 0.4 seconds. Popliteal valve reflux was unchanged. Postoperative APG showed that the VFI_{90} had been nearly normalized at 2.3 ml/s. Other values were essentially unchanged from preoperative levels.

Question 3

Which of the following is *not* true?

A. Postoperative DVT (30 day) is relatively rare after valve reconstruction procedures for correction of "primary" valve reflux.

B. Arm swelling occurs infrequently after axillary vein harvest for valve reconstruction.

C. Valve reconstruction is contraindicated in post-thrombotic veins.

D. Saphenous vein ablation can be safely undertaken in chronic deep venous obstruction (secondary saphenous varix).

E. In combined obstruction/reflux, stent placement to correct the obstruction alone often results in healing of stasis ulceration.

Commentary

The differential diagnosis of venous ulcers includes ischemic ulcers, diabetic foot ulcers, ulcers related to vasculitis from hypertension or other causes, ulcers related to connective tissue disorders (rheumatoid arthritis, scleroderma, etc.), neuropathic ulcers, Marjolin's ulcer, and numerous other conditions that are clinically quite rare. Popliteal *vein* (not artery) entrapment is a rare cause of venous ulcers [1]. The clinical features of venous ulcers are so characteristic and obvious that a positive diagnosis can be made on the basis of clinical examination alone in all but a few cases. When doubt exists, or when combined pathologies are suspected, a punch biopsy of the skin should be performed without hesitation to clarify the situation. Relevant testing for specific connective tissue, immunological or hematological conditions may be required in some cases. Venous ulcers are differentiated quite easily from arterial (ischemic) ulcers in most instances. The former are indolent and recurring with episodes of healing and breakdown and are generally confined to the gaiter area of the leg. In contrast, the arterial ulcer is progressive without periods of remission and has a wider distribution in the leg

with characteristic gangrenous or ischemic appearance devoid of granulation tissue and covered with necrotic tissue. There is seldom the surrounding hyperpigmentation or dermatitis that occurs so commonly with venous ulcers. Palpable pedal pulses virtually rule out ulcers of ischemic origin, with the notable exception of diabetic foot ulcers and less common entities in which vasculitis or small-vessel disease is often implicated (e.g. collagen disorders such as scleroderma and rheumatoid arthritis). It is usually possible, however, to narrow down the possibilities by a combination of clinical features (history, appearance and location of the ulcer), skin biopsy, and specific testing directed toward suspected non-venous pathology. Ankle/arm arterial index and toe pressure measurements may be required in some cases to clarify the issue. Because of their wide prevalence, venous ulcers can and do occur in combination with the other pathologies listed above. To establish the presence of venous ulcers in concert with other non-venous pathology, it is necessary to confirm that significant reflux is present based on venous duplex examination and venous hemodynamic tests such as ambulatory venous pressure measurement and/or air plethysmography. In combined arterial/venous ulcers, treatment should be directed initially towards improving arterial perfusion.

However obvious the diagnosis, patients with venous ulcers should be evaluated through a detailed assessment protocol to assess severity and form a base for later outcome assessment. Use of CEAP classification [2] and venous clinical severity scoring [3] provides a standardized format to accomplish this. Quality-of-life assessment methodologies [4] in venous disease have been validated and provide a way for outcome assessment from the patient's perspective. **[Q1: C]**

Many patients with chronic venous insufficiency will not volunteer information such as relief of leg pain with leg elevation and stocking use, night leg cramps and restless legs, or their developed habit of sleeping with the leg elevated at night, unless specifically asked. Perhaps because of the chronicity of the condition, these details have become an integral part of their daily lives and may not be mentioned as complaints without direct questioning. Even potentially important information, such as previous attacks of cellulitis or "phlebitis" that occurred years or decades ago and required hospitalization and a period of anticoagulant treatment may not be forthcoming unless specifically inquired, because the patient has forgotten the episode or does not consider it relevant to their current condition. Besides solidifying the diagnosis of venous ulcer, such information may be important in narrowing down the differential diagnosis in doubtful cases or combined pathologies. For example, ischemic rest pain at night is often relieved by hanging the leg over the side of the bed at night, whereas patients with venous pain seldom resort to this practice. Pain of claudication (arterial or venous) worsens with ambulation, whereas patients with limb pain from venous reflux have often learned to "walk off" their nocturnal pain. Venous claudication is estimated to occur in about 15 percent of patients with chronic venous insufficiency. Climbing up stairs is particularly difficult for these patients. Pain out of proportion to clinical signs is a characteristic of deep venous pathology. Pain, nocturnal leg cramps or restless legs may be the only clinical feature(s) in some patients. Recording the level of pain preoperatively by a visual analogue scale [5] is a simple reliable tool in severity assessment. The type and frequency of analgesic use (narcotic, non-narcotic, non-steroidal) is also useful. Past and current list of medications, particularly estrogen-type hormones and anticoagulants/platelet inhibitors, are relevant parts of the history and useful information in future management.

Limb swelling is a frequent manifestation of venous disease. It is hard to quantify by examination except in very gross terms. Plethysmographic techniques including the commonly used limb circumference measurement are unreliable as swelling is quite variable during the day with the extent of orthostasis. Patients' own perception of limb swelling is strongly influenced by the degree of accompanying pain. Patients themselves may not be aware of swelling obvious to the examiner if painless; conversely, even mild swelling when painful, may be rated as severe by the patient. For these reasons, quantification of swelling either by history or by examination is subject to considerable variance and error. Although some clinical features are described as unique to lymphedema in texts, differentiation of venous from lymphatic swellings on clinical grounds alone is generally not possible. Furthermore, the two pathologies frequently coexist. Lymphatic dysfunction appears to be secondary to venous obstruction in many cases; relief of venous obstruction can reverse the lymphatic dysfunction [6]. A thorough venous investigation is essential even when lymphoscintigraphy is abnormal.

The investigation of venous ulcers is directed toward positive establishment of venous etiology, identification of regional pathology, and assessment of hemodynamic severity. Hypercoagulability work-up provides guidance to the institution of anticoagulation, its duration and intensity. Duplex examination has replaced venography as the primary investigation for both screening and definitive assessment of chronic venous insufficiency. Overall accuracy of duplex ultrasound is superior to that of descending venography in the assessment of reflux [7, 8]. Duplex examination in the erect position yields more accurate results than does examination in the sitting or recumbent position [9]. Quick inflation/deflation cuffs with pressures set for various levels provide for standardized compression maneuvers and allow measurement of valve closure times; reflux is present when these exceed threshold values for the various valve stations. Disappointingly, valve closure times do not correlate with clinical or hemodynamic severity of reflux [10] and cannot be used in a quantitative way as originally hoped. The size and location of perforators can be assessed by duplex and is superior to physical examination. Patency of venous structures can be confirmed positively and post-thrombotic changes can be identified. Despite evolving refinement, duplex remains a largely qualitative morphologic technique.

Descending venography can document reflux through valve stations. The best results are obtained when the test is performed in the near-erect position with standardized Valsalva maneuver [11]. Comparison with duplex has led to the realization that the test, though sensitive, is not very specific. Descending venography is easily combined with transfemoral ascending venogram for assessment of the iliac veins, which may not visualize adequately by pedal injections of contrast. Even transfemoral venogram is only about 50 percent sensitive for detection of iliac vein obstructions [12]. Intravascular ultrasound (IVUS) is the gold standard for assessment of iliac veins for stent placement [13].

Ambulatory venous pressure is a global test of venous function. About 25 percent of patients with venous stasis ulceration have normal ambulatory venous pressure measurement parameters. Factors other than venous reflux, such as compliance, ejection fraction and arterial inflow, affect ambulatory venous pressure [14]. The latter factors are often abnormal in patients with chronic deep venous insufficiency. Consequently, ambulatory venous pressure often improves after valve reconstruction surgery but complete normalization is less frequent [15]. Measurement of ambulatory venous pressure via the dorsal

foot vein has been believed to accurately reflect deep venous pressure changes with calf exercise. Recent data throw considerable doubt on this long-held assumption [16].

Air-plethysmography is a non-invasive test of calf venous pump and can be used to assess surgical outcome [17]. Residual volume correlates with ambulatory venous pressure. However, venous filling index (VFI_{90}) has been a more consistent index of reflux with normalization after corrective surgery [18, 19].

Venous endothelial injury that occurs with deep venous surgery takes about 6 weeks to heal [20]. Patients should be anticoagulated adequately during this vulnerable period. With proper management, the thromboembolic complication rate is surprisingly low [21]. Patients who have suffered from previous bouts of thromboembolism and those with known hypercoagulable abnormalities are under increased risk of recurrent thrombosis and are candidates for long-term or even permanent anticoagulation. **[Q2: A]**

Once thought a rarity, primary deep venous reflux comprises about 30–40 percent of all deep venous reflux in centers active in deep venous reconstruction. Differentiating "primary" deep venous reflux from secondary or post-thrombotic reflux is problematic. The presentation and clinical features may be similar. Negative history for prior DVT may be unreliable as some thromboses are silent; and others might have been overlooked ascribing limb pain to trauma or orthopedic surgery that initiated it. Preoperative venography is a poor guide, and surgical exploration of the valve station is often the final arbiter [22]. Some patients with primary reflux develop actual distal thrombosis that can be recurrent. Correction of proximal reflux in this group of patients may alleviate these recurrent symptoms

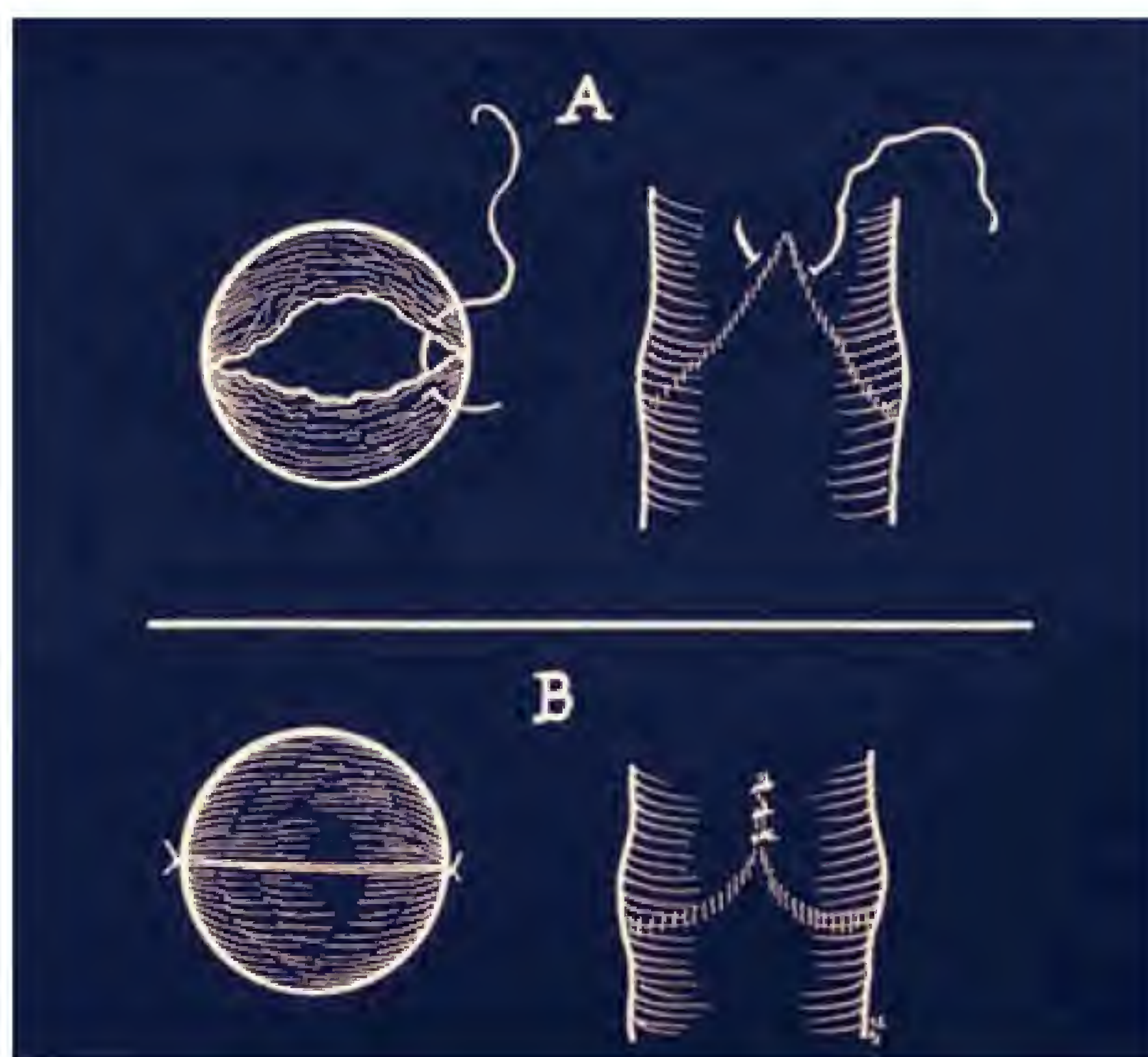


Fig. 44.1. Technique of transcommissural valvuloplasty. Transluminal sutures are placed from outside without opening the vein; they traverse the valve attachment lines, taking a bite of the free edge of each redundant valve cusp. When tied down, the valve attachment lines are brought together while tightening the valve cusps.

[23]. Conversely, deep venous thrombosis initiates by unknown mechanisms eventual development of reflux in adjacent and remote valve stations [24].

Correction of primary deep venous reflux by internal valvuloplasty was first described by Kistner in 1964. Subsequently, he described an external technique as well. A variety of open and closed techniques (Fig. 44.1) for correction of primary and post-thrombotic deep venous reflux are currently in use [25, 26]. The internal valvuloplasty technique has provided excellent results [21, 23, 27–29] and remains the standard (Fig. 44.2). The newer techniques provide a wider choice that may be more appropriate in certain circumstances, and yield clinical results similar to the original internal technique [21, 30]. Direct valvuloplasty may be feasible in some cases of post-thrombotic reflux where the valves have escaped destruction [22, 31]. Axillary vein valve transfer is the standard commonly used for correction of post-thrombotic reflux. It can be used with some modifications even in trabeculated veins with surprisingly good long-term patency and clinical success [32]. Arm swelling after axillary vein harvest is rare. **[Q3: C]**

The recent introduction of vein stent technology has decreased the number of valve reconstructions in our institution. Post-thrombotic syndrome is known to be due to a combination of obstruction and reflux in the majority of patients [33]. Use of intravascular ultrasound (IVUS) has shown that Cockett's syndrome (alias May–Thurner syndrome) is surprisingly frequent in "primary" reflux as well [34]. Correction of the obstructive component with stent placement in thrombotic and non-thrombotic cases results in excellent relief of pain and swelling and the patency rate is quite good [35]. Even totally occluded iliac veins can be successfully recanalized and stented [36]. Venous stasis ulcers are generally believed to be the result of reflux, not obstruction. Yet this relatively simple percutaneous outpatient stent technique results in healing of about 60 percent of venous stasis ulceration [37], even when the associated reflux remains uncorrected. Iliac vein stent placement is

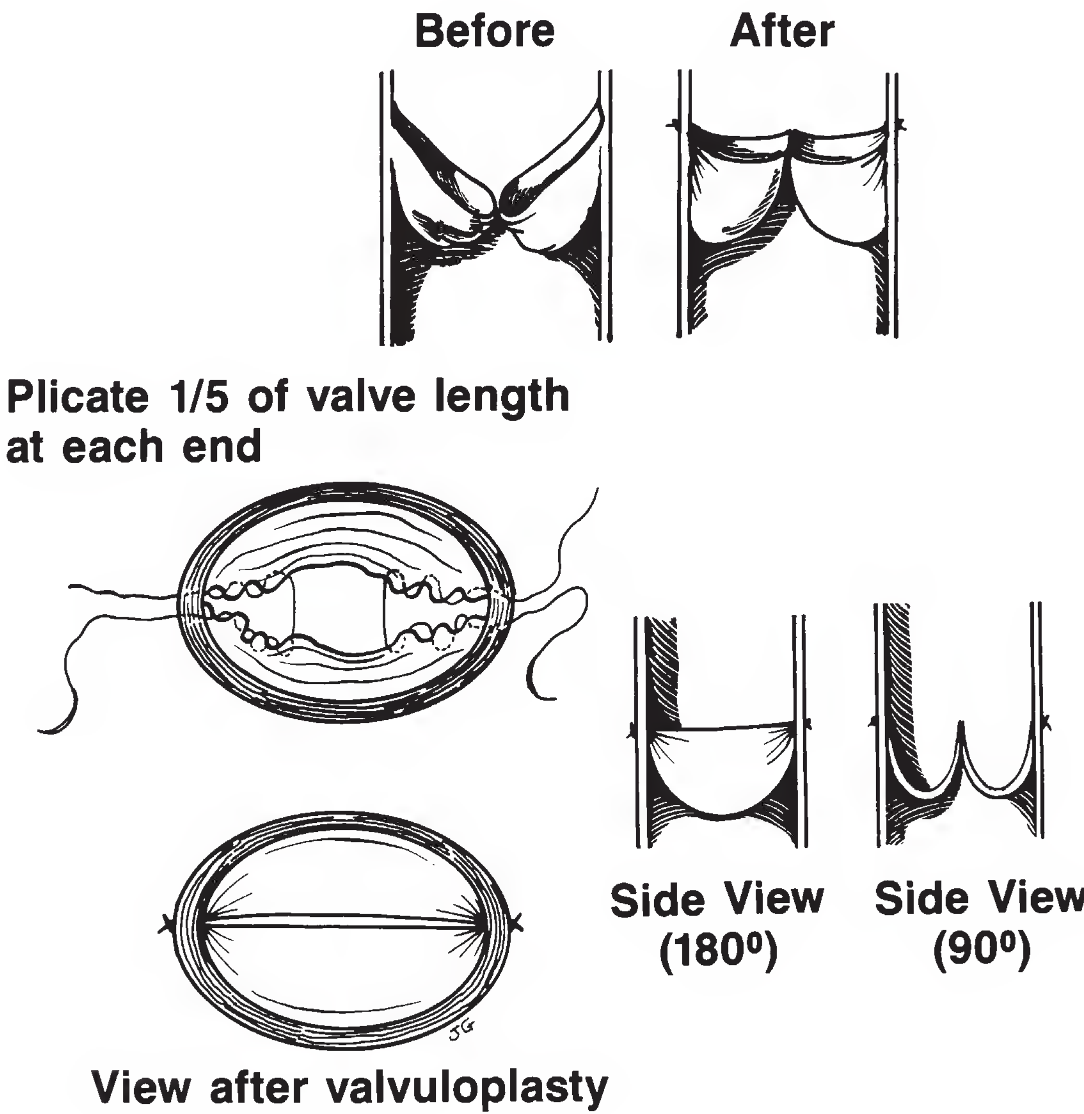


Fig. 44.2. Internal valvuloplasty technique.

currently the first choice in combined obstruction/reflux, and does not preclude later open surgery such as valve reconstruction or venous bypass if the stent were to fail. It is often combined with percutaneous laser ablation of the saphenous vein when reflexive. Saphenous ablation can be carried out safely, even in the presence of chronic deep venous obstruction (secondary varix) [38].

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45. Venous Ulcers Associated with Superficial Venous Insufficiency

Gudmundur Danielsson and Bo Eklöf

A 59-year-old female secretary was referred for evaluation and treatment of a non-healing painful ulcer on the medial aspect of her right lower leg. The ulcer had been recurrent almost every year for the past 9 years, often healing during the winter season. She had since early childhood been overweight (currently 87 kg, 170 cm, body mass index 30) and had difficulty in using compression stocking. She was otherwise healthy. She had two children, the first child born when she was 32 year of age and her second child 2 years later. After the birth of her second child she began to notice varicose veins on the lower leg on both sides and she often felt tiredness and heaviness in the leg in the afternoon. There was no history of deep venous thrombosis. She had been on birth control pills for 10 years and was currently on hormone replacement therapy because of severe postmenopausal symptoms. She had been treated at a local dermatological clinic for the past 2 years and was now being evaluated by a vascular surgeon. Clinical evaluation showed that she had 5 × 5 cm well-granulated ulceration above the right median malleolus which was surrounded by brownish leathery skin. She had slight swelling of the right leg with large varicosities below the knee. The left leg had large varicosities below the knee but no swelling or skin changes. Doppler examination revealed clear reflux in the groin that could be followed over both great saphenous veins (GSV) down the thigh. A possible minimal reflux was also noted in the popliteal fossa on the right side, although it was difficult to confirm this when the Doppler examination was repeated. Foot arteries were palpable on the dorsum of the foot on both sides.

Question 1

What should be the next step in this patient evaluation?

A. Measurement of ankle-brachial index.

B. Duplex ultrasound scanning of the venous system.

C. Plethysmography.

D. Ascending phlebography.

E. Biopsy of the ulcer.

Doppler measurement revealed a normal ankle/brachial index with systolic blood pressure 130 in both legs and right arm. Duplex ultrasound scanning of the venous system performed with the patient in 60° reversed-Trendelenburg position, using pneumatic cuff with automatic inflation/release on the lower leg to evaluate the reflux, showed bilateral reflux in the GSV, from the common femoral vein down to below knee, as well as two incompetent perforator veins on the medial aspect of the right calf with a diameter of 4 mm. The diameter of the GSV at the groin was 12 mm on the right side and 9 mm on the left side. The reflux time exceeded 4 s in both GSV, with peak reverse flow velocity more than 30 cm/s. Reflux less than 0.5 s was noted in the lesser saphenous vein on right side. No reflux was present in the deep veins except for minimal reflux in the common femoral vein with reflux duration of approximately 1 s on the right side. There were no signs of post-thrombotic changes.

Question 2

How should this patient be classified?

A. Leg ulcer.

B. Varicose ulcer.

C. C6,S,Ep,As,p,d,Pr.

D. C2,3,4b,5,6,S,Ep,As,p,d,Pr2,3,11,18.

The patient was classified according to the CEAP (clinical, (a)etiological, anatomical, pathophysiological) classification based on history and results of duplex ultrasound.

Question 3

Which of the following is not regarded as a risk factor for venous ulcer?

A. Diabetes.

B. Essential hypertension.

C. Smoking.

D. Overweight.

E. Resistance to activated protein C.

Question 4

What would be appropriate management for the right leg in this patient?

A. Conservative treatment with below-knee compression bandage, rest and leg elevation.

B. High ligation and stripping of GSV to below knee, with local extirpation of varicose veins.

C. High ligation of GSV with extirpation of varicose veins.

D. Obliteration of GSV using laser or radiofrequency heating with local extirpation of varicose veins.

E. Sclerotherapy with or without foam.

Question 5

How should the incompetent perforator veins be managed?

A. Subfascial endoscopic perforator surgery (SEPS).

B. Ligation through Linton–Cockett incisions.

C. Disregard them.

D. Ligation through small skin incisions.

E. Duplex-guided sclerotherapy.

Question 6

How should the left leg be managed?

A. Observation.

B. Sclerotherapy.

C. High ligation and stripping of GSV and local extirpation of varicose veins.

D. Obliteration of GSV using laser or radiofrequency heating and local extirpation of varicose veins.

The patient was treated with four-layer compression therapy until the operation day, which was postponed for 4 months. The ulcer and the swelling both decreased during this period; the ulcer measured 2×2 cm the day before operation. Both the right and the left leg GSV were treated with the closure method using radiofrequency derived heating, and varicose veins on the lower leg were extirpated through multiple small incisions. Intraoperative duplex ultrasound scanning revealed that both GSV were occluded with no sign of reflux and the deep veins were patent with no sign of deep venous thrombosis. No specific treatment was performed for the incompetent perforator veins. The patient was discharged the same day after uneventful postoperative recovery and was scheduled for new duplex ultrasound scanning after 2 and 7 days. The postoperative duplex ultrasound scanning was normal, with no sign of deep venous thrombosis, and the remnant of GSV was occluded. The patient continued with four-layer bandaging and went back to work on the fifth day after operation. The ulcer was healed at the last visit, which was 4 weeks later. Treatment with compression stocking during the daytime was planned for another 6 months.

Commentary

Investigation of both the arterial and the venous system is mandatory in cases of non-healing ulcer on the leg. Although Doppler examination had only revealed a clear reflux in GSV it is worthwhile to continue with duplex ultrasound scanning as deep venous incompetence and post-thrombotic changes can otherwise be overlooked. This is especially important when reflux is noted at the back of the knee where it is difficult with certainty to differentiate between deep venous reflux in the popliteal vein and reflux in the lesser saphenous vein. Although the history (no claudication or rest pain, no diabetes) and the location of the ulcer (medial aspect of lower leg) strongly suggest a venous ulcer, sometimes an arterial component is also present that might reduce the ability of the ulcer to heal. Palpable pulse on the dorsum of the foot (dorsalis pedis) or behind the medial malleolus (posterior tibial artery), as was evident in this case, almost rules out an arterial component. Although plethysmography can estimate the overall venous function it is not mandatory as a first line of investigation. Obtaining an ascending phlebography is also not necessary as it does not add any information that duplex ultrasound scanning does not provide and it is also an invasive method with the risk of complications. Non-healing ulcer with unusual appearance should be considered for other aetiology and investigated with biopsy in the early stage of evaluation. **[Q1: A, B]**

The old concept that the majority of venous ulcers are due to previous deep venous thrombosis [1, 2] has been altered during the last 20 years when duplex ultrasound studies have shown the importance of primary reflux in all venous segments [3–7]. Superficial venous incompetence is often noted to be the sole pathology in patients presenting with non-healing venous ulcer [8]. Formerly the venous ulcer was often judged as being related to a post-thrombotic condition without any objective diagnosis. Because of the benign course of varicose veins in the majority of patients with superficial venous incompetence, the need for thorough evaluation is often neglected. Formerly used classifications of chronic venous disease used the term varicose ulcer if varicose veins were present, or post-thrombotic ulcer if they were less evident or if there was a previous history of deep venous thrombosis. The importance of classification, based on findings from duplex ultrasound scanning, has become more evident during the last decades as treatment and prognosis is largely dependent on the background history and the results of clinical investigation. CEAP (clinical, (a)etiological, anatomical, pathophysiological) classification has gained more acceptance as the “gold standard” for classifying all aspects of venous pathology such as clinical class, aetiological background, anatomical distribution and pathophysiological findings (Table 45.1). There is a clear correlation between the CEAP clinical class and the venous function as measured by plethysmography (foot volumetry), indicating that the clinical classification has a realistic meaning concerning the functional evaluation of venous disease. The duration of reflux in venous segments, on the other hand, does not correlate with clinical class, but the peak reverse flow velocity is significantly higher in patients with skin changes/ulcer (C4–C6) [9]. The basic part of CEAP indicates the highest clinical class (C6, active venous ulcer) and the anatomical distribution in superficial, perforator or deep system (As, p, d) with reflux (Pr). S is added behind clinical class to indicate that the patient is symptomatic. The basic classification is sufficient for most clinical doctors. **[Q2: C, D]** The detailed version of CEAP is used when more information is needed as in longitudinal studies comparing treatment alternatives

Table 45.1. CEAP classification

Clinical classification
C0: no visible or palpable signs of venous disease
C1: telangiectases or reticular veins
C2: varicose veins
C3: oedema
C4a: pigmentation and/or eczema
C4b: lipodermatosclerosis and/or atrophie blanche
C5: healed venous ulcer
C6: active venous ulcer
S: symptoms including ache, pain, tightness, skin irritation, heaviness, muscle cramps, as well as other complaints attributable to venous dysfunction
A: asymptomatic
Aetiological classification
Ec: congenital
Ep: primary
Es: secondary (post-thrombotic)
En: no venous aetiology identified
Anatomic classification
As: superficial veins
Ap: perforator veins
Ad: deep veins
An: no venous location identified
Pathophysiological classification
Pr: reflux
Po: obstruction
Pr,o: reflux and obstruction
Pn: no venous pathophysiology identifiable

(Table 45.2). For more detailed information regarding the disease and its effect on daily life it is possible to use a venous severity scoring system [11]. Venous severity scoring is used as a complement to the CEAP classification (Fig. 45.1). Some medical conditions are clearly a risk factor for venous ulcer while others are less important. Venous ulcers are overrepresented in patients with diabetes although it is not clear if it is the venous pathology or if it is the diabetic microangiopathy that is the reason for this. Neither essential hypertension nor smoking is a proven risk factor for venous ulcer. The prevalence of varicose veins is increased in overweight individuals but the role of obesity is less clear when it comes to the risk of developing skin changes or ulcer. The apparent association between overweight and varicose veins in women suggests that it is a risk factor even in the more severe form of chronic venous disease [12–14]. In a consecutive series of 272 patients with chronic venous disease investigated with duplex ultrasound scanning, 58 per cent of patients with healed or open ulcer (C5–C6) had body mass index >30 kg/m^2 (obese) as compared to 15 per cent of those with varicose veins but without skin changes or ulcer [15]. **[Q3: A, B, C]** Most thrombophilic conditions are risk factors for deep venous thrombosis and venous ulceration, as is resistance to activated protein C [16]. The prevalence of thrombophilia is high in patients with venous ulceration despite no history or duplex ultrasound findings of deep venous thrombosis [17].

Table 45.2. Advanced CEAP

Same as basic CEAP with the addition that any of 18 named venous segments can be utilised as locators for venous pathology.
Superficial veins:
1. telangiectases/reticular veins
2. great saphenous vein above knee
3. great saphenous vein below knee
4. small saphenous vein
5. non-saphenous veins
Deep veins:
6. inferior vena cava
7. common iliac vein
8. internal iliac vein
9. external iliac vein
10. pelvic: gonadal, broad ligament veins, other
11. common femoral vein
12. deep femoral vein
13. femoral vein
14. popliteal vein
15. crural: anterior tibial, posterior tibial, peroneal veins (all paired)
16. muscular: gastrocnemial, soleal veins, other
17. perforating veins, thigh
18. perforating veins, calf

Surgical treatment is mandatory in cases of isolated superficial incompetence as the likelihood of ulcer recurrence otherwise will remain high. Conservative treatment alone with below-knee compression had not been successful in keeping the ulcer healed, but it is important to continue with compression therapy while the ulcer is open and for some time after operation. Four-layer bandage is effective in healing venous ulcer [18]. High ligation with stripping of the GSV down to below the knee, with local extirpation of varicose veins, is the method of choice. It decreases the risk of ulcer recurrence and has a low incidence of nerve damage to the saphenous nerve. Stripping of the vein from the groin to the ankle increases the risk of damage to the saphenous nerve (5 per cent versus 29 per cent), although the recurrence rate is still the same [19]. Just doing high ligation without stripping the vein is less feasible as the recurrence rate is significantly higher [20]. Other promising methods for ablation of the refluxing GSV have emerged recently and might become the methods of choice in the future. As the diameter of the GSV was less than 15 mm it was possible to use the radiofrequency closure method to obliterate the vein. The main advantage of using less invasive methods is increased patient satisfaction, as the recovery time after operation has been reported to be shorter. Follow-up time up to 5 years with the radiofrequency method indicates that the method is durable. The long-term results after ablation of GSV using laser technique or foam sclerotherapy are still unknown. **[Q4: B, D]**

The varicose veins on the lower leg are dealt with by using multiple stab incisions and bringing them out using hooks. The cosmetic results are better and the risk of nerve damage is less. Care should be taken not to operate close to the ulcer area as healing problems and infection are more common if the incisions are made in damaged skin.

First Name Sara

Last Name Anderson

Leg included ○ Left ◉ Right

Visit day 12/12/2004

History | Symptom and sign | CEAP | Score | Surgery

Clinical class	Active ulcer
Etiologic class	Primary
Anatomic class	SDP
Pathophysiologic class	Reflux

C0	0
C1	0
C2	1
C3	1
C4	1
C5	1
C6	0

Qualifying comments

Clinical class C2,3,4b,5,6,S-Ep-As,d,p-Pr2,3,11,18

Venous Segmental Disease Score

Reflux

LSV	◉ 0 ○ 0.5
GSV	○ 0 ◉ 1
Perforating Thigh	◉ 0 ○ 0.5
Perforating Calf	○ 0 ◉ 1
Calf veins, multiple	◉ 0 ○ 2
Popliteal	◉ 0 ○ 2
Femoral	◉ 0 ○ 1
Deep femoral	◉ 0 ○ 1
Common femoral and above	○ 0 ◉ 1
	3

Obstruction

Obstruction GSV	◉ 0 ○ 1
Obstruction calf vein multiple	◉ 0 ○ 1
Obstruction popliteal vein	◉ 0 ○ 2
Obstruction FV	◉ 0 ○ 1
Obstruction PFV	◉ 0 ○ 1
Obstruction CFV	◉ 0 ○ 2
Obstruction iliac vein	◉ 0 ○ 1
Obstruction IVC	◉ 0 ○ 1
	0.0

diameter of perforator 4.0 (mm)

adjacent to ulcer ◉ Yes ○ No

Venous segmental disease score 3.0

History | Sympto and sign | CEAP | Score | Surgery

Clinicalal score

Pain	3
Varico seveins*	2
Venous** edema	1
Skin pigmentation	1
Inflammation	1
Indu ration	1
Tolal no. ulcer***	1
Active ulceration, duration	1
Active ulcer size	3
Compressive therapy	1
Total clinical score	15

0=Absent
1=Mild
2=Moderate
3=Severe

Qualifying comments

Disability score

Disability score 2

0=A symptomatic
1=Symptomatic,work without support
2=Can carry out usual* activitie only with compression and/or limbelevation
3=Unable to carry out us ual* activities even with compression and/or limb elevation

*Usual activities=patients activities before onset of disbility from venous disease

Severity score (C#A#D)

20

Severity score mean 20

Fig. 45.1. Venous severity scoring is used as a complement to the CEAP classification.

Even though the role of surgery in venous ulcer disease has been unclear [21], a recently reported randomised controlled study comparing surgery with compression therapy, to compression therapy alone, could clearly show a significantly lower recurrence rate in the surgically treated group [22]. Altogether 500 patients with open or recently healed ulcer (6 months) were included in the study. The healing

rate was similar during the study period, but 12-month ulcer recurrence rates were significantly reduced in the surgically treated group or 12 per cent, compared to the compression-only group where the ulcer recurrence rate was 28 per cent.

The pathophysiology behind venous ulcer is mainly reflux as opposed to obstruction or occlusion. In a study on a consecutive series of 98 legs with an open venous ulcer, 85 per cent of the extremities had some form of superficial venous incompetence that might be treated with a simple operation on the superficial venous system. Axial reflux in the superficial (great saphenous vein) or the deep veins (femoral down to popliteal level) was present in 79 per cent of the legs [23]. Incompetent perforator veins and their role in chronic venous disease have been debated for years [24]. Incompetent perforator veins have been implicated as an important factor in the formation and recurrence of venous ulcers. This view is mainly based on clinical reports of excellent ulcer healing following the interruption of incompetent perforators. There is substantial evidence that subfascial endoscopic perforator surgery (SEPS) is effective in interrupting perforator veins, and it can be done without major wound complications that were often seen after the open subfascial Linton procedure [25–28]. Also, the ulcer healing rate after venous procedures that included SEPS has been satisfying [25, 26]. Patients undergoing surgery for incompetent perforator veins almost always have surgery simultaneously on the superficial venous system and therefore it is difficult to judge the actual contribution of the incompetent perforator to the venous dysfunction. There is also evidence that reflux-eliminating surgery on one part of the venous system can abolish reflux in another part [29–31]. Operations on superficial veins have been shown to eliminate concomitant reflux in perforators [32]. Disregarding the incompetent perforator veins in patients with superficial venous incompetence seems therefore to be appropriate. The low incidence of isolated perforator incompetence in patients with active venous ulcer does indicate that they are less important than previously thought [8]. The main indication (although not proven yet) for treating them is in patients with primary venous incompetence with recurrent ulceration despite optimal treatment of the superficial venous incompetence. The method of choice for treatment is then SEPS, mainly because of the low risk of wound complication. The use of sclerotherapy for the purpose of obliterating perforators is still under evaluation although the technique seems to be promising. **[Q5: A, C]**

The indication for treating varicose veins in legs without skin changes or ulcer is less clear. The decision of recommending treatment for asymptomatic legs with varicose veins has to be judged individually; often it is the patient's preference that will decide. The cosmetic results of sclerotherapy on local varicose veins are poor if the refluxing GSV is left in place. The risk of future problems with skin changes or ulcer is increased when axial reflux is present in the GSV, as was the case with this patient, and that might be a sufficient reason to recommend even surgery for the asymptomatic left leg. A simultaneous operation on both legs in an otherwise healthy person does not seem to add any risk to the operation. If a catheter-based ablation is used to obliterate the GSV it is feasible to treat both legs at the same time as one catheter can then be used to treat both legs as the catheter is expensive. **[Q6: A, C, D]**

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46a. Iliofemoral Venous Thrombosis

William P. Paaske

A 72-year-old man was admitted in the late evening because of a turgid, white, painful left leg. Over the course of 4 months, he had lost 8 kg of weight (from 82 to 74 kg); his height was 175 cm. There were general symptoms, such as tiredness, slight nausea, lack of appetite and increasing apathy. Over the last 12 h, he had been increasingly confused and aggressive. He had been bedridden for 3 weeks but had refused to see a doctor. There was no history of psychiatric disease, focal cerebrovascular events, ischaemic heart disease, hypertension, intermittent claudication, or venous insufficiency. He had been smoking about 20 cigarettes a day since he was 14 years old, and for many years he had had slight functional dyspnoea, but otherwise no pulmonary symptoms. Stools had been light yellow to grey/white for the last week. His renal function had never been examined, and it had not been noticed whether he had passed urine in the last 24 h. Diazepam was the only medication. The history was provided by his wife, who had called the ambulance. Medical records were not available.

The patient was confused, with delusions; he was intermittently agitated and possibly psychotic, but he could be calmed down. He looked chronically ill, slightly emaciated, possibly anaemic and dehydrated. Temperature and blood pressure (arms) were normal. There was tachycardia with a regular rhythm. The abdomen was slightly distended, but there was neither a palpable mass nor peritoneal reactions. Digital rectal exploration was unremarkable. The right leg was normal with distal pulses.

The left leg had diffuse swelling from the groin to the toes; there was moderate pallor, and no visible varicosities when standing. There was no evidence of superficial thrombophlebitis. Minor venous collaterals were noticed in the groin and just above the inguinal ligament. During palpation over the deep femoral veins, the patient groaned and became increasingly aggressive. The consistency of the calf muscle groups was increased with tenderness but not woody. There was less floppiness of the left leg muscles compared with those of the right. Spontaneous dorsiflexion of the foot was noticed, but sensory function could not be assessed due to lack of patient cooperation.

The quality of the pulses in the groin and knee was good, and the pulse in the dorsal pedal artery was possibly present. Capillary filling in the pulp of the toes

could not be assessed. The plantar pallor did not increase during elevation, but discrete colour change was noticed at the back of the foot during post-elevation dependency.
Bladder catheterisation did not produce urine. Electrocardiogram (ECG) was normal apart from a rate of 114 bpm.

Question 1

What is the most likely diagnosis?

A. Thrombosis of the crural veins.

B. Thrombosis of the femoral veins.

C. Thrombosis of the iliac and femoral veins.

D. Thrombosis of the superficial femoral artery.

E. Thrombosis of the external iliac and superficial femoral arteries.

Blood samples were taken, and the patient was admitted.

Question 2

Which investigation should be ordered and carried out at once?

A. Intravenous arteriography.

B. Intra-arterial arteriography.

C. Ascending phlebography.

D. Colour duplex sonography.

E. Plethysmography.

Due to an unusually large number of emergency admissions, the patient had to wait several hours before he could be examined by colour duplex sonography and have his chest X-ray taken. An hour before the scheduled time for these examinations (8 h after admission), and before the results of the blood tests were available, the patient deteriorated and the pulse rose further. His temperature was now 38.9°C. He had become increasingly agitated and complained of severe pain in the left leg.

Haemorrhagic bullae developed on the back of the foot and around the medial ankle, and the skin of the rest of the foot and the distal calf showed numerous petechiae. There was increased swelling, and the colour of the leg turned deeply cyanotic, even during elevation. The tips of all the toes were black. A weak pulse could be felt in the femoral artery in the groin, but distal pulses were absent. The consistency of the muscle groups of the thigh as well as the lower leg was clearly increased, and the patient suffered severe pain when femoral muscles were assessed

by compression. He did not react to pain induced by pinching the skin from the knee and distally. At this point, the right leg exhibited slight but definite swelling, and the skin was beginning to become cyanotic. Pulses could still be felt in the right groin and popliteal artery, but pedal pulses had disappeared.

Question 3

What is/are the common name(s) for this clinical presentation?

A. Iliofemoral venous thrombosis.

B. Iliofemoral phlebothrombosis.

C. Phlegmasia alba dolens.

D. Phlegmasia cerulea dolens.

E. Venous gangrene.

Question 4

What ideally should have been done, and what should be done at this stage at 4 a.m. on the basis of this clinical presentation and with the additional information provided above?

At this point, the results of the blood tests taken in the emergency room became available: they showed anaemia with haemoconcentration, thrombocytopenia and electrolyte derangements; S-creatinine was 410 mmol/l, and the leucocyte count was 14 times above the upper normal limit. The large amount of fluid sequestered in the gangrenous left leg may account for part of the haemoconcentration.

Question 5

Would you consider a surgical thrombectomy at this stage? If so, how would you perform it?

The situation was deemed hopeless and beyond medical therapy. The patient was given intravenous morphine to relieve the pain, and he died 13 h after admission.

Commentary

The tentative diagnosis at admission was acute left-sided iliofemoral venous thrombosis with the clinical picture of phlegmasia alba dolens. **[Q1: C]** It was highly probable – but not proven – that this bedridden patient suffered from active malignant disease with secondary venous thrombosis. His general appearance in connection with the specific signs and symptoms, including apparent lack of urine production, indicated a disaster in progression. The association of cancer and deep venous thrombosis is well established [1]. If a malignancy is definitely diagnosed, or

suspected with a high degree of certainty, disseminated and/or in an advanced stage where expected residual lifespan is very short, then ultrasonically or phlebographically verified iliofemoral venous thrombosis with venous gangrene (ischaemic venous thrombosis) must be interpreted as one of the signs indicating imminent termination of life, and treatment (medical as well as operative) is generally contraindicated, including on compassionate grounds.

With unreliable, rudimentary or uncertain information, it is essential that diagnosis is established not merely as soon as possible but at once; it is not acceptable to wait several hours for the diagnostic test, colour duplex sonography. **[Q2: D]** The patient should be taken immediately to the ultrasound examination room and, if necessary, scanned by the surgeon. **[Q4]**

In our case, the colour duplex sonography after a phase of phlegmasia cerulea dolens progressing to manifest venous gangrene on the left side and phlegmasia cerulea dolens in development on the right, showed bilateral thrombosis of both femoral and iliac veins in addition to thrombosis of the inferior vena cava up to and above the renal veins, which explained the lack of urine production. **[Q3: E]**

Certain assessment algorithms have been devised for the management of this condition [2], but the remotest suspicion of deep venous thrombosis should result either in colour duplex sonography or ascending phlebography (with digital subtraction technique), or in magnetic resonance venography with gadolinium enhancement plus T1 images (bull's eye sign) [3], if available, in patients with renal impairment or allergy to angiographic contrast media. Some centres have the option of computed tomographic (CT) venography (possibly with spiral/slip-ring technique), which has the additional advantage of being able to visualise extravascular morphology. Plethysmography (strain gauge, impedance, air, etc.) must be considered obsolete for precise diagnosis; isotope uptake tests have generally been disappointing and should be avoided. Both legs, rather than only the symptomatic leg, should be examined in all patients. In patients with coexisting arterial insufficiency of the lower extremities, the diagnosis can be even more difficult, so investigations should be performed at a lower level of clinical suspicion. Where phlegmasia cerulea dolens is surmised, or where venous gangrene is apparent, then one of these examinations must be performed without delay. If pulmonary embolism is suspected, then lung scintigraphy, pulmonary angiography or magnetic resonance or CT scanning of the pulmonary arteries should be performed.

Treatment aims to prevent or decrease further thrombus formation or propagation, to reduce or stop acute (pulmonary embolism) and chronic (post-thrombotic syndrome) complications, and to reduce pain.

In principle, the thrombus can be reduced, or removed, by chemical or mechanical means. A fresh thrombus is generally less adherent than an old thrombus. The preferred treatment of iliofemoral venous thrombosis is heparin administered for 3–4 days as a continued intravenous infusion (high-dose heparin) concomitant with oral phenprocoumon, dicoumarol or warfarin, which should be given for a further 3–6 months [4]. This regime probably has no influence on the development of chronic post-thrombotic syndrome.

Thrombolysis with streptokinase, urokinase or recombinant tissue plasminogen activator (rt-PA) can be attempted for thrombi less than about 10 days old under close monitoring with repeated colour duplex sonography examinations [5–9]. Although many series have been published, the effect on pulmonary embolism is dubious, and the long-term clinical results of properly conducted studies are still poorly documented.

Interruption of the venous system between the thrombus and the heart prevents pulmonary embolism and may be considered in highly selected cases; it is performed by partial or complete occlusion of the inferior caval vein by either open surgery [10] or deployment of temporary or permanent caval filters [11], of which several types are commercially available. The long-term outcomes of both techniques are not clear. The incidence of filter complications – early as well as late – is not negligible.

Once again, as in many other aspects in the treatment of venous disease, there are widely diverging opinions as to the place of surgical thrombectomy of iliofemoral venous thrombosis with or without construction of an arteriovenous fistula. In pregnancy or during puerperium, surgical thrombectomy should not be attempted [12]. A balanced view, based on the available literature, would be that it may be a possibility that could be considered in limb-threatening phlegmasia cerulea dolens [13–17].

The operation is performed with the supine patient in the reversed Trendelenburg position (legs down), and under general anaesthesia with continuous positive airway pressure. The femoral veins, which may bulge with thrombus, and arteries are exposed in the groin by a longitudinal incision. After slings have been applied, a longitudinal phlebotomy is made, and a venous Fogarty catheter is advanced towards the heart. The balloon is inflated, and the catheter is withdrawn together with the thrombus. The procedure is repeated until no more thrombus is delivered. The leg is now elevated, and by manual compression (possibly followed by compressing bandage, e.g. Esmarch's), one aims to remove the thrombus within the leg. The phlebotomy is then closed. The patient should have the leg elevated until mobilisation after a few days. Thrombectomy often results in incomplete clot removal and recurrence [18].

In certain centres, the contralateral groin vessels are also routinely exposed, a Fogarty catheter is introduced into the common femoral vein, and the tip is positioned in the upper part of the inferior caval vein. The balloon of this catheter is insufflated during the manoeuvres on the contralateral side, and it is retracted with inflated balloon after each of the Fogarty thrombectomy procedures. The aim is to retract fragments of thrombus and avoid (additional) pulmonary embolism.

Some surgeons advocate construction of an arteriovenous fistula in addition to the surgical thrombectomy. The great saphenous vein is transected as appropriate below the saphenofemoral junction, and the distal part of the proximal segment is anastomosed to an arteriotomy in the common femoral artery. The aim is to increase blood flow, thereby reducing the risk of recurrent thrombus formation, in the proximal part of the femoral vein and veins proximal to that. **[Q5]**

Although the extremity with phlegmasia cerulea dolens may look very bad indeed, a conservative approach is warranted (careful monitoring, elevation of the leg, heparin, fluid replacement). If systemic symptoms or signs occur, or if the situation deteriorates into manifest venous gangrene, then amputation must be performed without delay.

Operative treatment of chronic iliofemoral venous thrombosis and its sequelae, notably post-thrombotic syndrome, remains controversial. Reports with various reconstructions, e.g. with polytetrafluoroethylene (PTFE), remain anecdotal [19].

Endovascular treatment options are emergent, some in combination with open surgery (hybrid procedures). Combined application of transcutaneous thrombectomy devices, balloon angioplasty, stenting etc. with thrombolysis may lead to a

new level of therapeutic aggressiveness [20], but proper scientific documentation is so far not available, and these new developments must be considered experimental.

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46b. Iliofemoral Deep Venous Thrombosis (During Pregnancy)

Anthony J. Comerota

A 24-year-old female who was 32 weeks pregnant presented to the emergency department at 7 p.m. with a swollen, painful left lower extremity. Her left leg had become progressively more symptomatic during the past 48 hours. During the past 24 hours, she began feeling lethargic, had slight shortness of breath, and began to experience right chest discomfort with deep breathing.

Upon physical examination, her heart rate was 106/min, respiratory rate was 18/min, and blood pressure was 112/70. Her lungs were clear, and her abdomen was appropriate for her gestational age.

She had a swollen left leg from the foot to the inguinal ligament, which had a bluish hue. She had pain upon palpation of the left femoral vein. Her arterial examination was normal.

A venous duplex was ordered and scheduled to be performed in approximately 3 hours.

Question 1

At this point, what would be your next course of action?

A. Obtain an immediate ventilation/perfusion lung scan.

B. Perform a venogram.

C. Start intravenous heparin at 75 mg/kg bolus followed by a continuous infusion at 15 mg/kg/hour; or, an injection of subcutaneous enoxaparin at 1 mg/kg.

D. Maintain the patient at bed rest until the duplex is completed. If the duplex confirms deep vein thrombosis (DVT), begin treatment with heparin.

E. Perform an echocardiogram.

The patient had an intravenous line started and a bolus of unfractionated heparin was given, followed by a continuous infusion. Four hours later, the venous duplex examination demonstrated venous thrombosis in the posterior tibial vein, popliteal

vein, femoral vein, proximal great saphenous vein, common femoral vein, and external iliac vein to the visible limit of the examination. The veins of the right lower extremity were normal. The patient asks, "What can I expect if treated with continued anticoagulation?"

Question 2

You tell the patient that she has iliofemoral and infrainguinal deep vein thrombosis, and that with continued anticoagulation:

A. She will do much better following delivery if she remains anticoagulated for 1 year.

B. She faces a 15–40 percent likelihood of venous claudication at 5 years.

C. She faces a 90 percent likelihood of venous insufficiency and 15 percent likelihood of venous ulceration.

D. It is difficult to predict the natural consequences of her disease.

Question 3

This patient's father has long suffered with post-thrombotic chronic venous insufficiency, and she expresses a strong desire to avoid post-thrombotic complications. However, she does not want to accept the risks of bleeding associated with thrombolytic therapy; therefore, she asks for your treatment recommendation. Your best recommendation to this patient would be:

A. Intravenous heparin for 5 days, followed by oral anticoagulation with a warfarin compound.

B. Heparin (unfractionated or low-molecular-weight) until the delivery, followed by warfarin anticoagulation.

C. Rheolytic thrombectomy.

D. Catheter-directed thrombolysis.

E. Operative venous thrombectomy.

Because of her painful lower extremity and her concern for post-thrombotic complications, the patient requested that the thrombus be removed. She was reluctant to accept the potential bleeding complications of catheter-directed thrombolysis, and the attending radiologist was reluctant to treat with catheter-directed lysis. Therefore, venous thrombectomy was planned.

Question 4

The next appropriate step is:

A. Obtain a ventilation/perfusion scan or spiral CT scan of the chest to evaluate for suspected pulmonary embolism.

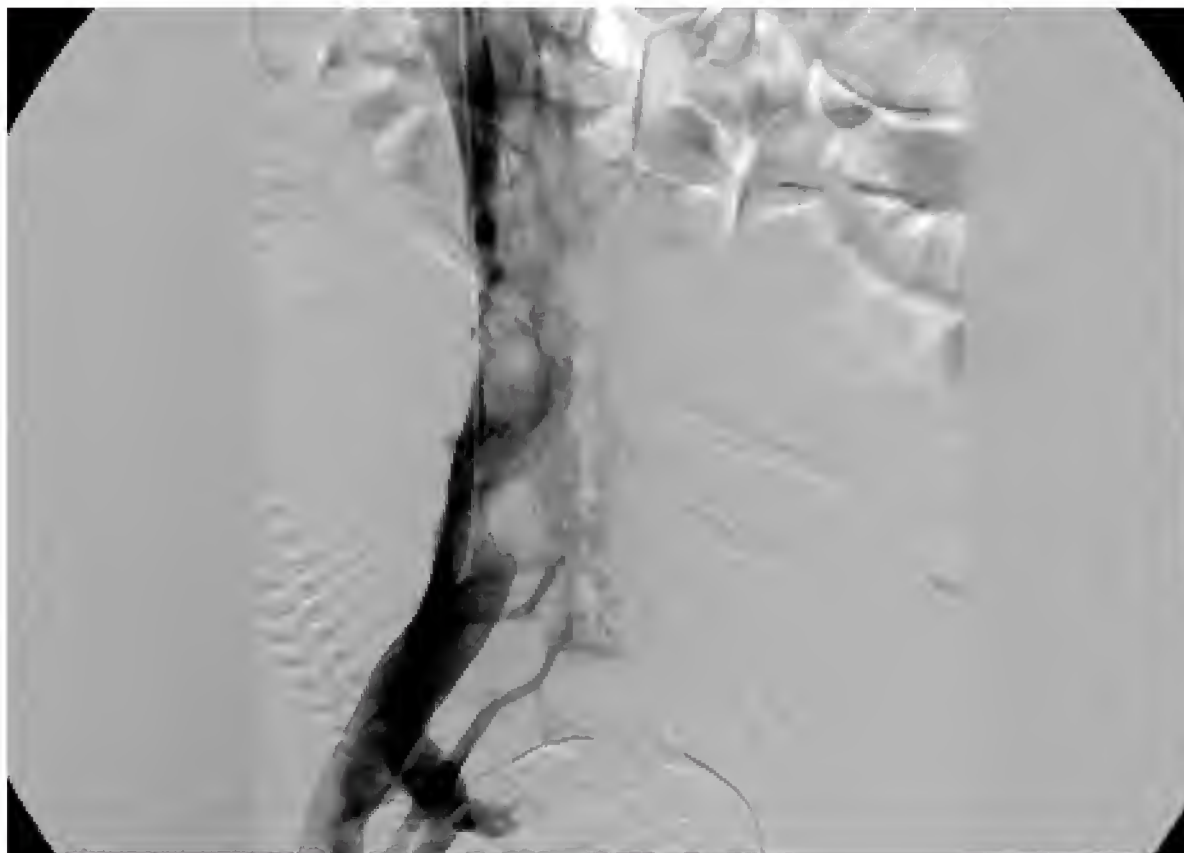


Fig. 46b.1. A contralateral iliocavagram demonstrates a large volume of nonocclusive thrombus in the vena cava. Note fetal skeleton in normal position.

B. Obtain a contralateral iliocavagram prior to taking the patient to the operating room.

C. Take the patient directly to the operating room and perform the procedure in order to avoid progressive deterioration.

D. Anticoagulate overnight and proceed with operative thrombectomy the next day.

The patient was anticoagulated with intravenous heparin overnight. The next morning a contralateral iliocavagram was performed (Fig 46b.1) prior to taking the patient to the operating room. A large volume of nonocclusive thrombus was found throughout the infrarenal vena cava.

Question 5

In light of the findings on the cavagram, what is the best next step?

A. Abandon operative venous thrombectomy and anticoagulate.

B. Perform an AngioJet mechanical thrombectomy of the vena cava and iliofemoral venous system.

C. Perform a pulmonary arteriogram to confirm/exclude pulmonary embolism.

D. Obtain an echocardiogram.

E. Insert a suprarenal vena caval filter and proceed with venous thrombectomy under fluoroscopic guidance.

The patient was presumed to have had a pulmonary embolism. A echocardiogram failed to show right ventricular dysfunction, an enlarged right ventricle, tricuspid insufficiency, or elevated pulmonary artery pressures. Because of the



Fig. 46b.2. X-ray demonstrates suprarenal vena caval filter in proper position.

potential risk of dislodging nonocclusive thrombus during the venous thrombectomy, a removable suprarenal vena caval filter was inserted (Fig. 46b.2).

Question 6

Important considerations during thrombectomy include:

A. Shield the fetus from all X-ray exposure.

B. Perform the venous thrombectomy under fluoroscopic guidance.

C. Monitor the fetus throughout the procedure.

D. Let the nonocclusive thrombus in the vena cava remain undisturbed and perform a thrombectomy of the iliofemoral venous system only.

The patient was taken to the operating room for a venous thrombectomy with fluoroscopic guidance and fetal monitoring. A cut-down was performed on the left common femoral and femoral veins, with exposure of the saphenofemoral junction. A longitudinal venotomy was performed at the level of the saphenofemoral junction, followed by protrusion of a large amount of acute thrombus. The leg was raised and a tight rubber bandage applied with minimal extrusion of the infrainguinal thrombus. Attempts to pass a catheter from the inguinal ligament distally into the femoral vein and attempts to pass a guidewire distally were unsuccessful.

Question 7

The next appropriate step would be:

A. Perform iliofemoral and caval thrombectomy with AV fistula, leaving the infrainguinal thrombus.

B. Abandon thrombectomy and anticoagulate.

C. Perform an infrainguinal venous thrombectomy aided by a cut-down on the left posterior tibial vein.

A cut-down on the posterior tibial vein was performed. Following a posterior tibial venotomy, a no. 3 Fogarty catheter was passed upwards through the thrombosed venous system, exiting the common femoral venotomy. This catheter was used to guide a no. 4 Fogarty catheter distally through the venous valves by placing both catheter tips within a 14-gauge Silastic intravenous catheter sheath after the hub was amputated. Following a mechanical balloon catheter thrombectomy, the leg was flushed using a bulb syringe with a large volume of heparin/saline solution, which flushed additional thrombus from the common femoral venotomy. After clamping the femoral vein, the deep venous system was then filled with 300 ml of a dilute recombinant tissue plasminogen solution (6 mg rt-PA in 300 ml).

The iliofemoral and vena caval thrombectomy was performed under fluoroscopic guidance, filling the balloon with contrast to ensure that the suprarenal caval filter was not dislodged. After completing the thrombectomy, an operative iliocavagram was performed to assess the adequacy of thrombectomy and to ensure unobstructed venous drainage into the vena cava. An iliac vein stenosis was observed.

Question 8

The appropriate next step is:

A. Close the venotomy and anticoagulate, since a common iliac vein stenosis is frequently observed due to normal vascular anatomy.

B. Close the venotomy and perform an AV fistula.

C. Perform angioplasty and insert a self-expanding stent if recoil occurs.

D. Operatively expose the common iliac vein and perform an endovenectomy and transpose the vein above the right common iliac artery.

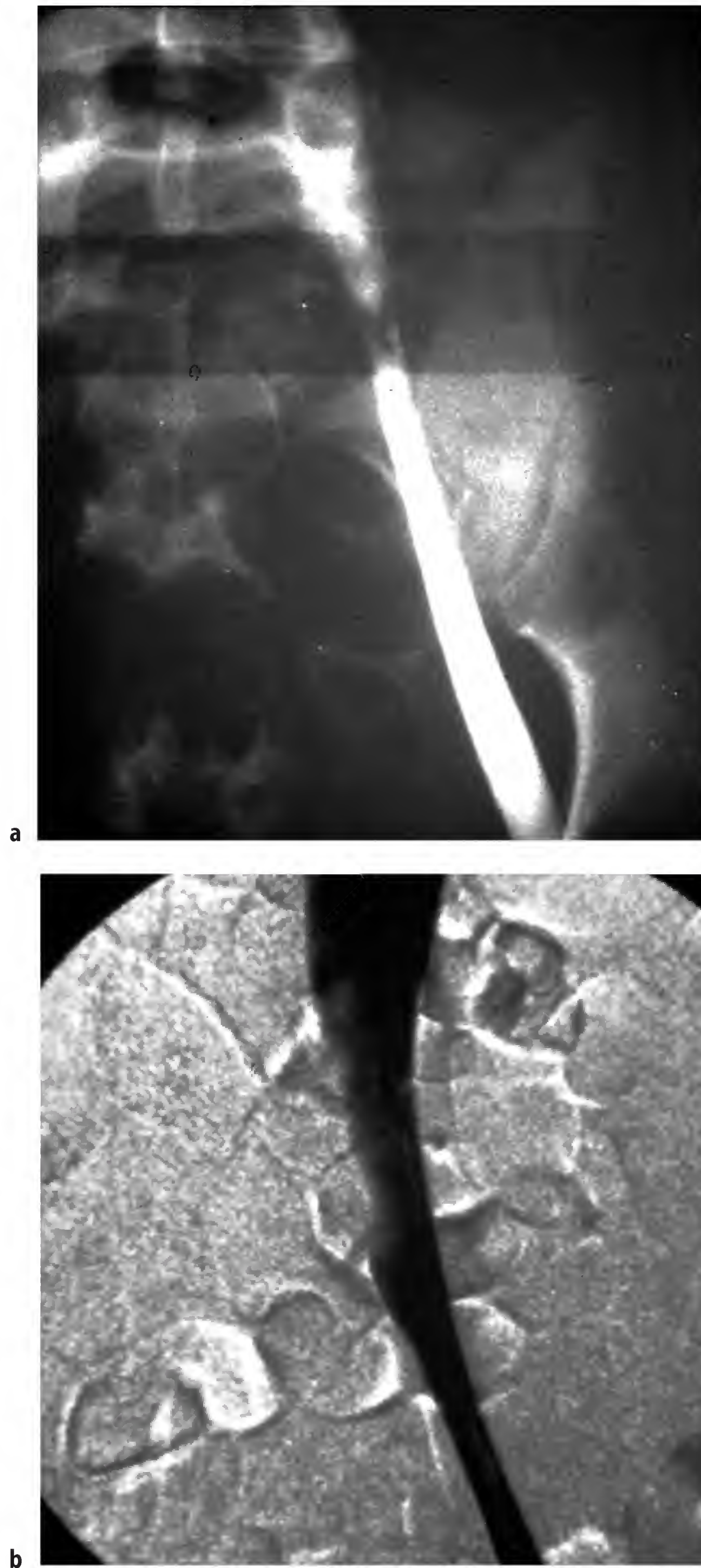


Fig. 46b.3. a A completion phlebogram following iliofemoral thrombectomy shows stenosis of the left common iliac vein. **b** Balloon dilation corrects the lesion without evidence of recoil, providing unobstructed venous drainage into the vena cava.

A balloon angioplasty catheter was placed into the lesion and an angioplasty performed. The iliac vein was dilated to 14 mm without evidence of recoil (Fig. 46b.3).

Question 9

Now that patency has been restored to the infrainguinal and iliofemoral venous systems, are there any additional techniques that can be performed to reduce risk of rethrombus?

A. An AV fistula, using the end of the proximal saphenous vein sewn to the side of the superficial femoral artery.

B. The saphenous vein should not be used for AV fistula, since it represents collateral drainage from the leg in the event of recurrent thrombosis.

C. Placement of a catheter into the posterior tibial vein for anticoagulation with unfractionated heparin.

D. Elevate the legs and avoid ambulation for the next 4–5 days.

E. Therapeutic anticoagulation.

An arteriovenous fistula (AVF) using the proximal saphenous vein anastomosed to the superficial femoral artery increases flow velocity through the iliofemoral venous system, reducing the risk of rethrombosis. A thrombectomy of the proximal great saphenous vein was required in this patient, as is often the case. Since the goal of the AVF is to increase venous blood flow velocity, the size of the anastomosis is limited to 3.5–4 mm in order to avoid a steal and avoid venous hypertension. A small piece of PTFE is wrapped around the saphenous AVF and looped with a 2-cm piece of O-Prolene, which is left in the subcutaneous tissue (Fig. 46b.4). This will serve as a guide should the AVF require closure. However, since the AVF is small, it is considered permanent and closure is not anticipated.

To further reduce the risk of rethrombosis, a heparin infusion catheter (pediatric feeding tube) is placed into the proximal posterior tibial vein and brought out through a separate stab wound adjacent to the lower leg incision. Infusing unfractionated heparin through this catheter to achieve a therapeutic PTT ensures a high concentration of heparin in the target vein, a concentration much higher than would be achieved if the patient was treated with standard intravenous anticoagulation through an arm vein. A monofilament suture is looped around the catheter in the posterior tibial vein and brought out through the skin and secured with a sterile button. This is used to occlude the vein after 5–6 days when the catheter is removed following full oral anticoagulation with warfarin. In the case of this pregnant patient, intravenous anticoagulation through the leg veins was maintained for 4 days, after which she was converted to subcutaneous enoxaparin at 1 mg/kg every 12 hours. The catheter was removed and the patient discharged. The patient was maintained on subcutaneous enoxaparin 1 mg/kg twice a day until she delivered a healthy baby 6 weeks later.

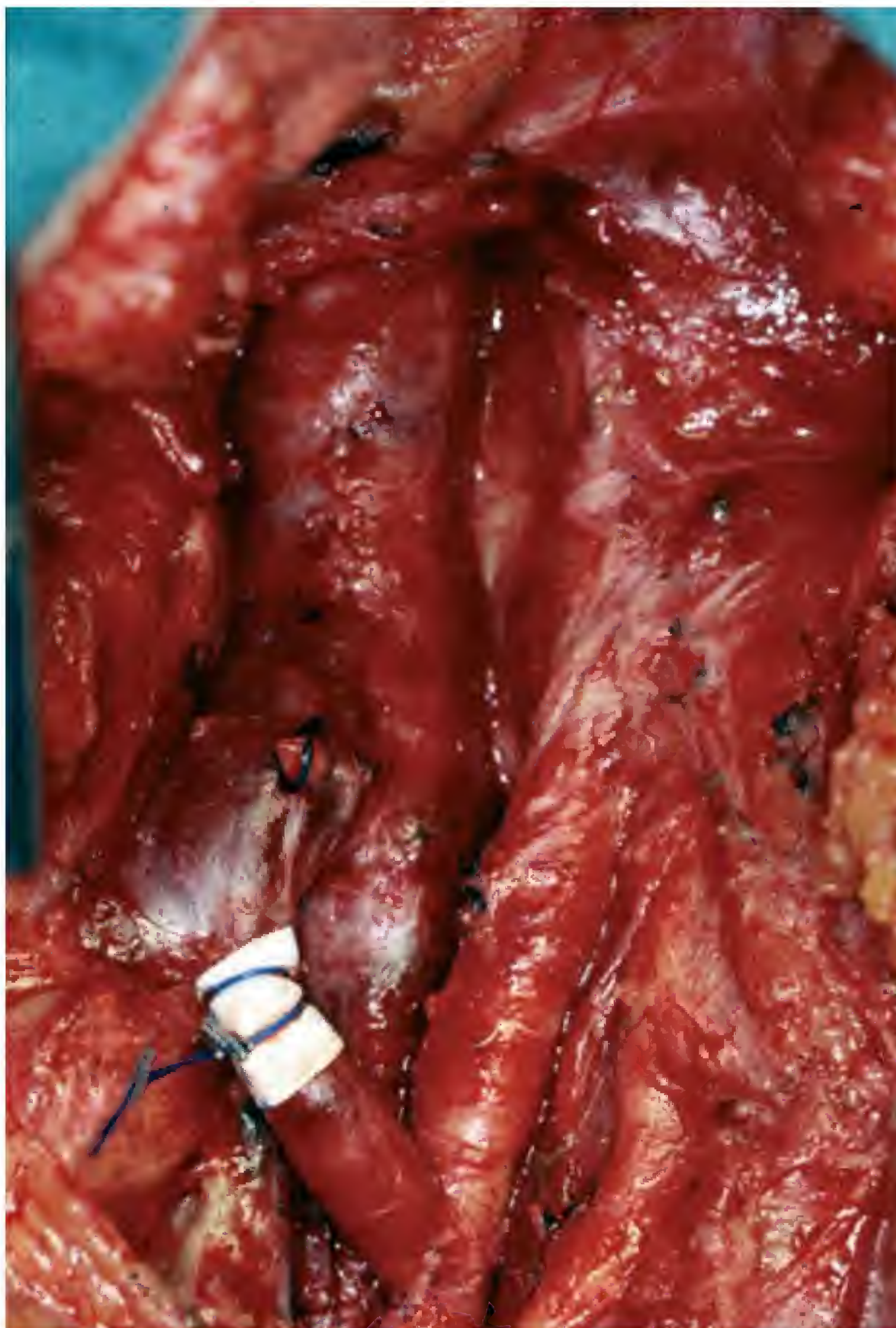


Fig. 46b.4. The construction of the arteriovenous fistula (AVF) using a large side branch of the great saphenous vein sutured end-side to the superficial femoral artery. Note sleeve of PTFE wrapped around the AVF and looped with a 2-cm piece of O-monofilament suture. The purpose of this is to assist in operative closure should obliteration of the AVF become necessary.

Question 10

The patient does not wish to breastfeed her baby. What is your best recommendation for ongoing therapy?

A. Six more weeks of Lovenox.

B. Oral anticoagulation for 6–12 months.

C. Patients' risk for recurrence is determined by the amount of residual thrombus. If there is no residual thrombus on venous duplex, additional anticoagulation is unnecessary.

D. Stop anticoagulation and start aspirin.

Question 11

What is your recommendation regarding a thrombophilia evaluation?

A. It is not necessary to perform an expensive thrombophilia evaluation since this was a DVT of pregnancy.

B. Defer the thrombophilia evaluation until after the patient discontinues anticoagulation.

C. Since this patient will be on indefinite anticoagulation, a thrombophilia evaluation is not necessary.

D. Perform tests not affected by anticoagulation and complete the evaluation after anticoagulation has been discontinued.

An abbreviated thrombophilia evaluation of: lupus anticoagulant, antiphospholipid/anticardiolipin antibody, factor V Leiden, prothrombin gene mutation, and homocysteine was negative. The remainder of the thrombophilia evaluation will be completed in 1–2 years, at which time it is anticipated that the patient's Coumadin will be discontinued.

Commentary

Although not recognized by the recent ACCP guidelines on antithrombotic therapy for venous thromboembolism [1], iliofemoral deep venous thrombosis represents a condition with a uniquely high incidence of post-thrombotic morbidity [2–4].

This patient's presentation was clinically consistent with iliofemoral deep venous thrombosis associated with a pulmonary embolism. The adventitia of the femoral vein is innervated with sensory nerves; therefore, pain on palpation of the femoral vein as a result of its distension is a frequent physical finding. The femoral vein distends as a result of the associated venous hypertension and thrombosis.

Patients presenting during off hours to the emergency department who are at high clinical risk of a venous thromboembolic condition should be anticoagulated **[Q1: C]** until a definitive diagnosis is made [1].A ventilation/perfusion (V/Q) lung scan is not performed in this patient because she is pregnant and the clinical probability of a pulmonary embolism is high. The likelihood of the venous duplex demonstrating acute DVT is also high; therefore, this patient's treatment is unlikely to be altered by the V/Q scan findings. There is also reluctance to expose the pregnant patient to a radioisotope. Standard ascending phlebography is not necessary, since the clinical presentation and venous duplex will establish the diagnosis with a high degree of accuracy. Once anticoagulation is established, it is not necessary and actually counterproductive to maintain the patient at bed rest [5]. An echocardiogram is advisable in all patients who have the diagnosis of pulmonary embolism to evaluate its impact on right ventricular function; however, it is not necessary in this patient to perform an "off hours" echocardiogram since the patient can be adequately treated until the next business day.

This patient's thrombus extends from the posterior tibial vein to the external iliac vein, as documented on venous duplex. The natural history of these patients is one

of significant post-thrombotic morbidity. **[Q2: B, C]** Akesson and colleagues [3] demonstrated that within 5 years of anticoagulation for iliofemoral deep venous thrombosis, 95 percent of patients had documented venous insufficiency, 15 percent had venous ulceration, and 15 percent suffered with venous claudication. Delis et al. [4] studied in greater detail a similar but larger cohort of patients with iliofemoral deep venous thrombosis and performed exercise testing. They demonstrated that 40 percent developed symptoms of venous claudication. While pregnancy is an induced hypercoagulable state, delivery of the present patient's child is not known to alter the natural history of the patient's acute venous thrombosis.

In order to reduce the high risk of post-thrombotic sequelae, a strategy of thrombus removal should be considered. Operative venous thrombectomy **[Q3: E]** is the best recommendation in light of the fact that the patient does not wish to face the additional risk of bleeding with thrombolytic therapy, and the radiologist is reluctant to treat the patient with catheter-directed thrombolysis. Rheolytic thrombectomy is in its early stages, and to date has not been shown to be effective by itself in the absence of incorporating a plasminogen activator [6]. Oral anticoagulation during pregnancy is not recommended. Although this patient is in her third trimester and warfarin embryotrophy is not a concern, the potential coagulopathy of the fetus due to its immature liver and potential fetal bleeding complications during delivery as a result of passage through the birth canal make oral anticoagulation inadvisable. Heparin anticoagulation until delivery followed by oral anticoagulation is commonly offered to these patients; however, their post-thrombotic morbidity is exceptionally high.

A decision was made to proceed with venous thrombectomy. Patients can be anticoagulated overnight and the operation performed the next business day. Venous thrombectomy does not need to be performed as an "emergency operation." **[Q4: B, D]** In all patients in whom a venous thrombectomy is performed, it is important to know the proximal extent of thrombus, particularly whether there is thrombus in the inferior vena cava. Therefore, a contralateral iliocavagram is performed prior to the iliofemoral venous thrombectomy. As mentioned earlier, it is assumed that this patient has had a pulmonary embolism and the radiation exposure of a CT scan or a V/Q scan is unnecessary, since their results are unlikely to change this patient's management. However, in the non-pregnant patient, a spiral CT scan of the chest, abdomen, and pelvis would be performed. The rationale for CT scanning is that approximately 50 percent of patients with proximal DVT will have an asymptomatic pulmonary embolism. Up to 25 percent of these patients will develop subsequent pulmonary symptoms [7]. When the symptoms surface during anticoagulation, the symptoms are often misinterpreted as "failure" of anticoagulation, when in reality it is the natural evolution of the patient's initially asymptomatic (undiagnosed) pulmonary embolism. The proximal extent of thrombus in the vena cava or iliac veins often can be identified, as well as screening for associated intra-abdominal, retroperitoneal, or pelvic pathology.

The patient was treated with anticoagulation overnight. Before going to the operating room a contralateral iliocavagram was performed. Information regarding the proximal extent of thrombus is particularly important, since the details of thrombus extension may alter the procedure. Nonocclusive thrombus in the vena cava is concerning because of its potential for fragmentation and embolization. This author believes that these patients should be protected against potential embolization during the procedure. This can be accomplished either with a suprarenal vena caval filter, as was inserted in this patient, since it was presumed that she already had suf-

fered a symptomatic pulmonary embolism. Alternatively, suprathrombus balloon occlusion during the caval thrombectomy can be performed. This patient also underwent a preoperative echocardiogram to evaluate the impact of her presumed pulmonary embolism on right ventricular function. Echocardiography should be performed in all patients with pulmonary embolism, since it is a predictor of chronic thromboembolic pulmonary hypertension, and patients who have right-sided abnormalities should be considered for thrombolytic therapy or mechanical thromboembolectomy. **[Q5: D, E]**

During the operative procedure, fluoroscopy is used to guide the placement of the balloon catheter so as not to dislodge the vena caval filter. Fluoroscopy is also used to assess the success of thrombectomy and to evaluate for underlying venous lesions and their correction (Fig. 46b.3). Since the fetus is very well developed, the risk to the fetus from modest X-ray exposure is low. Fetal monitoring is routinely performed throughout the procedure. The monitoring devices must be checked so as not to interfere with appropriate imaging of the venous system during the procedure. Shielding of the fetus would obscure the iliac veins and distal vena cava. **[Q6: B, C]**

Previous descriptions of iliofemoral venous thrombectomy focus only on the iliofemoral venous system. An occluded infrainguinal venous system reduces venous return through the thrombectomized iliofemoral veins, and leaves substantial thrombus burden infrainguinally with its resultant post-thrombotic sequelae. Current techniques of infrainguinal venous thrombectomy allow the procedure to be performed successfully following a cut-down on the posterior tibial vein. **[Q7: C]** Therefore, contemporary venous thrombectomy should be viewed much the same as arterial thrombectomy, that is, removing as much thrombus from the venous circulation as is physically and pharmacologically possible, correcting any underlying lesion, and perform mechanical and pharmacological maneuvers to avoid recurrent thrombosis.

An iliac venous stenosis observed on completion phlebography is common. Correcting the underlying iliac vein stenosis is considered an important part of the procedure (Fig. 46b.3). This is performed under fluoroscopic guidance and if recoil occurs, a self-expanding stent is used to maintain unobstructed venous drainage from the iliac venous system into the vena cava. **[Q8: C]** Direct endophlebectomy of the iliac vein lesion and transposition above the right common iliac artery is a large operation, which has been replaced by the relatively simple balloon dilation and stenting.

Following successful thrombectomy of the infrainguinal and iliofemoral venous systems and correction of any underlying iliac vein stenosis, prevention of recurrent thrombosis is paramount. There are mechanical and pharmacologic measures which, if used, minimize recurrence. These include the construction of a femoral AV fistula using the end of the transected proximal saphenous vein (or a large side branch) anastomosed to the side of the proximal superficial femoral artery (Fig. 46b.4). Frequently, the proximal saphenous vein must undergo a thrombectomy to restore its patency. The saphenous vein is not a collateral pathway of venous drainage for patients with iliofemoral venous thrombosis. On occasion, it may be a collateral drainage pathway for patients with infrainguinal DVT. Since the infrainguinal venous system had patency restored, that is not an issue in this patient. The AV fistula is constructed to increase venous velocity in the iliofemoral veins; however, it should not increase venous pressure. Limiting the size of the anastomosis to approximately 4 mm usually accomplishes this goal. Pressure monitoring of the common femoral vein before and after flow is initiated through the AVF is

important. If the venous pressure increases, one must suspect a proximal (iliac vein) stenosis or excessive flow through the AVF, either (or both) of which should be corrected.

An additional, effective adjunctive technique is the placement of a catheter into the posterior tibial vein, which is used to anticoagulate the patient with unfractionated heparin postoperatively. A pediatric feeding tube is inserted into the posterior tibial vein and brought out through a separate stab wound in the skin adjacent to the lower leg incision. This small catheter is used for postoperative anticoagulation with unfractionated heparin. Targeting a therapeutic PTT ensures a high concentration of heparin in the diseased vein, which should substantially reduce the risk of recurrence. In the author's experience, when these adjunctive techniques have been used, no patient has experienced rethrombosis. **[Q9: A, C, E]**

Following delivery, women can be anticoagulated with Coumadin, assuming they do not wish to breastfeed. Warfarin is excreted in the breast milk of women; therefore, those who breastfeed should not be taking warfarin compounds. Among the options, oral anticoagulation for 6–12 months is the most appropriate. **[Q10: B]** While it is true that residual thrombus increases the risk of recurrent thrombosis [8], it would be inappropriate to treat this patient with less than a full course of anticoagulation. Since this patient had extensive venous thrombosis and a positive family history, an underlying thrombophilia is suspected and the author would extend the duration of anticoagulation to 1 year or more.

A thrombophilia evaluation is appropriate in this patient. A complete thrombophilia evaluation cannot be performed while the patient is on anticoagulation, since antithrombin III, proteins C and S, and factor VIII will be affected. However, lupus anticoagulant, antiphospholipid antibody, factor V Leiden, prothrombin gene mutation, and homocysteine levels can be obtained during anticoagulation and, if positive, may play a role in the subsequent management of this patient. **[Q11: D]**

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47a. Management of Upper Extremity Lymphoedema with Microsurgical Lymphovenous Anastomosis

Corradino Campisi and Francesco Boccardo

A 59-year-old woman presented with an 8-year history of oedema of the left arm. Initially, the oedema appeared in the upper arm. The patient was treated with combined decongestive physiotherapy (manual and mechanical lymphatic drainage), bandaging and exercises three to four times over a 12-month period. Despite these measures, the oedema extended as far as the forearm and hand (Fig. 47a.1), and she had several episodes of erysipeloid lymphangitis. In the months preceding her admission, she also complained of episodes of lymphangitis and pain. There were no warts or wounds on the skin. Her past medical history included lumpectomy with axillary lymphadenectomy and radiotherapy for left breast cancer, although routine follow-up for breast cancer did not suggest any local recurrence.

Initially, the oedema had a rhizomelic location. It was hard to the touch without pitting oedema. There were no dystrophic or dyschromic skin lesions, except for signs of acute reticular erysipeloid lymphangitic attacks caused by Gram-positive *Staphylocci* infections promoted by lymph stasis. A lymphangioscintigram was performed, which showed features compatible with lymphatic circulatory impairment in the left arm (Fig. 47a.2). This was followed by lymphangio-magnetic resonance imaging (MRI) of the left arm and hemithorax, which showed no signs of locoregional relapse of breast cancer but confirmed lymph stasis, predominantly in the epifascial compartment. In addition, it showed dilated medial arm lymphatic collectors interrupted at the proximal third of the arm. Finally, echo-Doppler of the left subclavian and axillary venous axis was performed. This did not demonstrate any venous dysfunction. A diagnosis of chronic secondary lymphoedema of the left arm following breast cancer treatment was made.

Question 1

How do you classify lymphoedema?



Fig. 47a.1. Patient before treatment.

Question 2

Which of the following statements regarding the diagnosis of lymphoedema are correct?

A. Lymphangiography is currently the best diagnostic investigation for all kinds of lymphoedema.

B. The echo-Doppler investigation has an important role in determining the correct treatment for the patient.

C. Lymphangioscintigraphy is the most popular noninvasive first-line investigation for lymphoedema.

D. It is difficult to diagnose lymphoedema at an early stage.

E. Lymphangio-MRI offers precise morphological data on oedema distribution and topography of dilated lymphatic pathways, without requiring contrast.



Fig. 47a.2. Lymphangioscintigram before microsurgery.

The patient underwent microsurgical lymphatic-venous anastomoses in the proximal third of the volar surface of the left arm using an 8/0 nylon suture material (Fig. 47a.3).

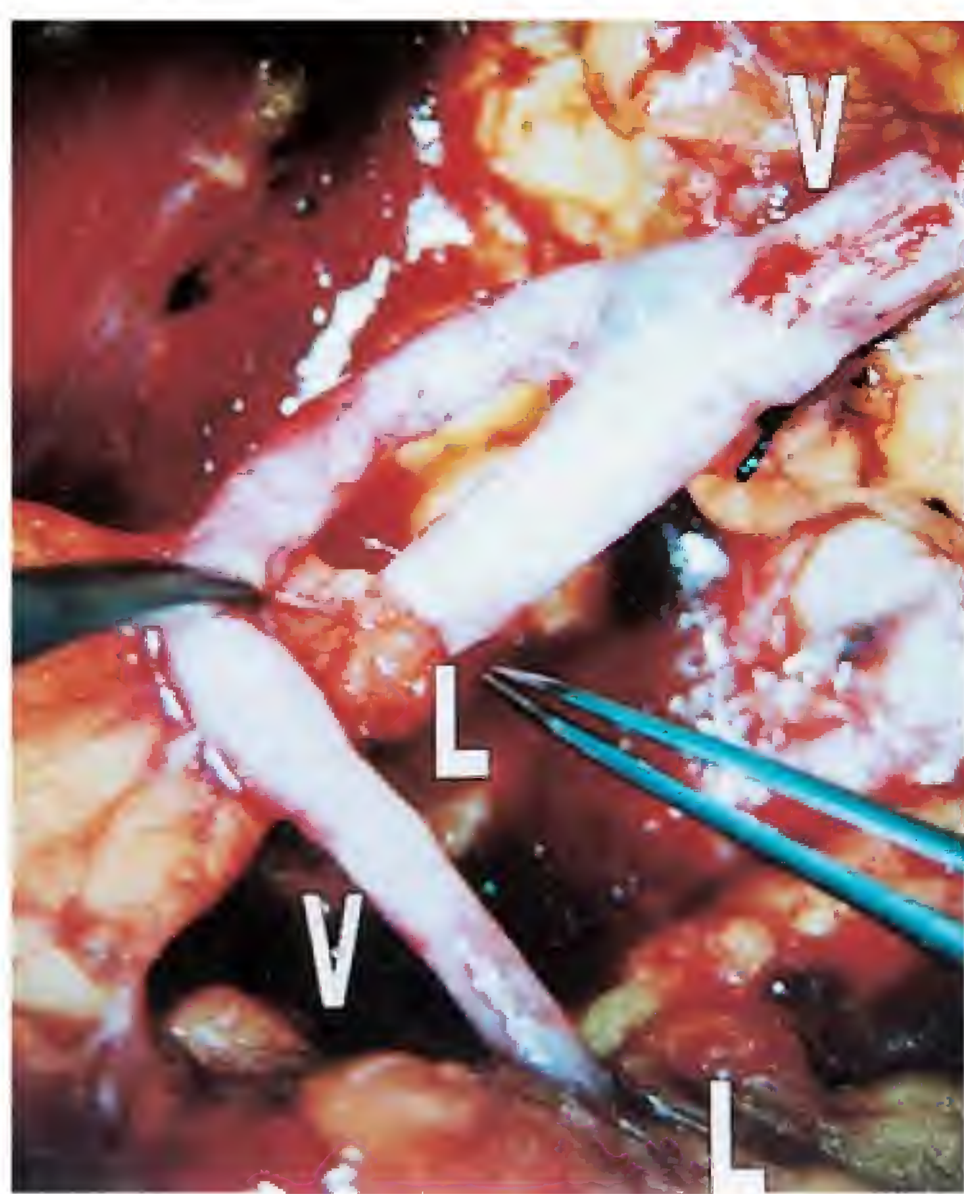


Fig. 47a.3. Lymphatic-venous anastomoses seen through the operating microscope (30¥).

Question 3

Which of the following statements regarding the management of lymphoedema are correct?

A. Microsurgery can reduce oedema in all patients, but the best outcome is seen in patients operated on in the second and third stages.

B. Proper elastic compression garments are an important adjunct to optimise long-term results.

C. Surgical intervention is not indicated in the advanced stages of lymphoedema.

D. Microsurgical lymphatic-venous anastomoses are used more frequently than reconstructive microsurgical methods.

E. Microsurgery cannot be applied in primary lymphoedema.

The postoperative recovery was uneventful. The patient was discharged home on the fifth postoperative day. The incidence of lymphangitic attacks decreased significantly.



Fig. 47a.4. Long-term clinical outcome after microsurgery.

A reduction in arm volume was seen within 3 days of the operation, and further improvements were observed at medium- and long-term follow-up, particularly between the first and the fifth years after surgery. From the fifth year onwards, the clinical condition of the arm remained stable with time, even more than 10 years after the operation (Fig. 47a.4). Lymphangioscintigraphy at this point demonstrated that the lymphatic-venous anastomoses were still patent (Fig. 47a.5).

Question 4

What are the long-term results of derivative and reconstructive microsurgery for lymphoedema?

Question 5

In what ways can secondary lymphoedema be prevented?

Commentary

Lymphoedema is a significant worldwide problem. It can be divided into primary and secondary forms. Primary lymphoedemas do not have any recognisable cause



Fig. 47a.5. Lymphangioscintigram performed after microsurgery shows the patency of the lymphatic-venous anastomoses more than 10 years after the operation.

(so-called idiopathic), although triggering aetiological factors can often be found. Lymphoedemas that present at birth (congenital) are included in this category. These can be hereditary-familial (Nonne–Milroy's disease), and are often associated with chromosomal abnormalities. Other primary lymphoedemas may have an early or late onset, which can be triggered by minor trauma, infection or surgery. In females, the predisposing factors are often thought to be alterations in neurohormonal status (neuroendocrine lymphoedema).

Primary lymphoedemas can also be due to lymphatic and/or lymphnodal dysplasia, hypoplasia or even hyperplasia with associated increased lymph production. Lymph nodes and/or lymphatics can be involved in abnormal lymph flow. In most cases of hypoplasia, lymph node involvement is demonstrated and leads to the progressive secondary alteration of lymphatic vessels. From pathophysiological and diagnostic points of view, this picture is practically the same as that seen with secondary lymphoedemas resulting from lymphadenectomy and/or radiotherapy [1].

Approximately 90 per cent of all primary lymphoedemas are characterised by hypodysplastic alterations involving lymph nodes and/or lymphatics. This is characterised by a diminished ability to form and activate a proper collateral circulation in response to trauma, infection and surgery. In a further 8–10% of primary lymphoedemas, an increased number and size of lymphatic collectors can be found, associated with structural lymphatic and lymph nodal dysplasia [2].

Disorders in lymphogenesis also play an important role from a lymphodynamic point of view. Hyperlymphogenesis may derive from pre-existing regional arterial-venous hyperstomies, arterial-venous fistulae (i.e. in Klippel–Trenaunay's disease) or related angiodysplasia. In contrast, reduced or absent production of lymph, agenesis, hypoplasia, or impaired permeability of the initial lymphatics is very rare, if not exceptional.

Finally, among lymphodynamic abnormalities, apart from insufficient lymph drainage along anatomically pre-established pathways, gravitational lymph and/or chylous reflux pathologies should also be mentioned. This top-to-bottom lymph backflow is caused by insufficient antigravitational structures, normally represented by valves, the reticular myoelastic layer of the lymphatic walls, and lymph node architecture (lymphoedemas and chyloedemas due to gravitational reflux).

The aetiology of secondary lymphoedemas can generally be identified in the patient's history or physical examination. This can be secondary to trauma, infection, inflammation, radiotherapy, surgery, paralysis or even neoplasia. Indeed, lymphatic filariasis is endemic in some tropical and subtropical areas of Asia, Africa and Latin America. However, secondary lymphoedemas often have some congenital predisposition. For instance, congenital wall-valve dysplasia of the lymphatics is always found in lymphatic filariasis. Similarly, arm lymphoedema secondary to breast carcinoma treatment occurs in 5–35% of cases, depending on whether axillary surgery is associated with radiotherapy [3]. However, it is more likely to occur when there is no deltoid pathway [4]. This lymphatic way drains the lymph coming from the arm directly into the supraclavicular lymph nodes, thus bypassing the axillary stations. With preventive lymphoscintigraphic studies, comparing the arm ipsilateral to the breast cancer site with the contralateral one, patients with a higher risk of developing secondary lymphoedema could be identified and could, theoretically, receive preventive therapeutic treatment. Based on these observations, Tosatti's

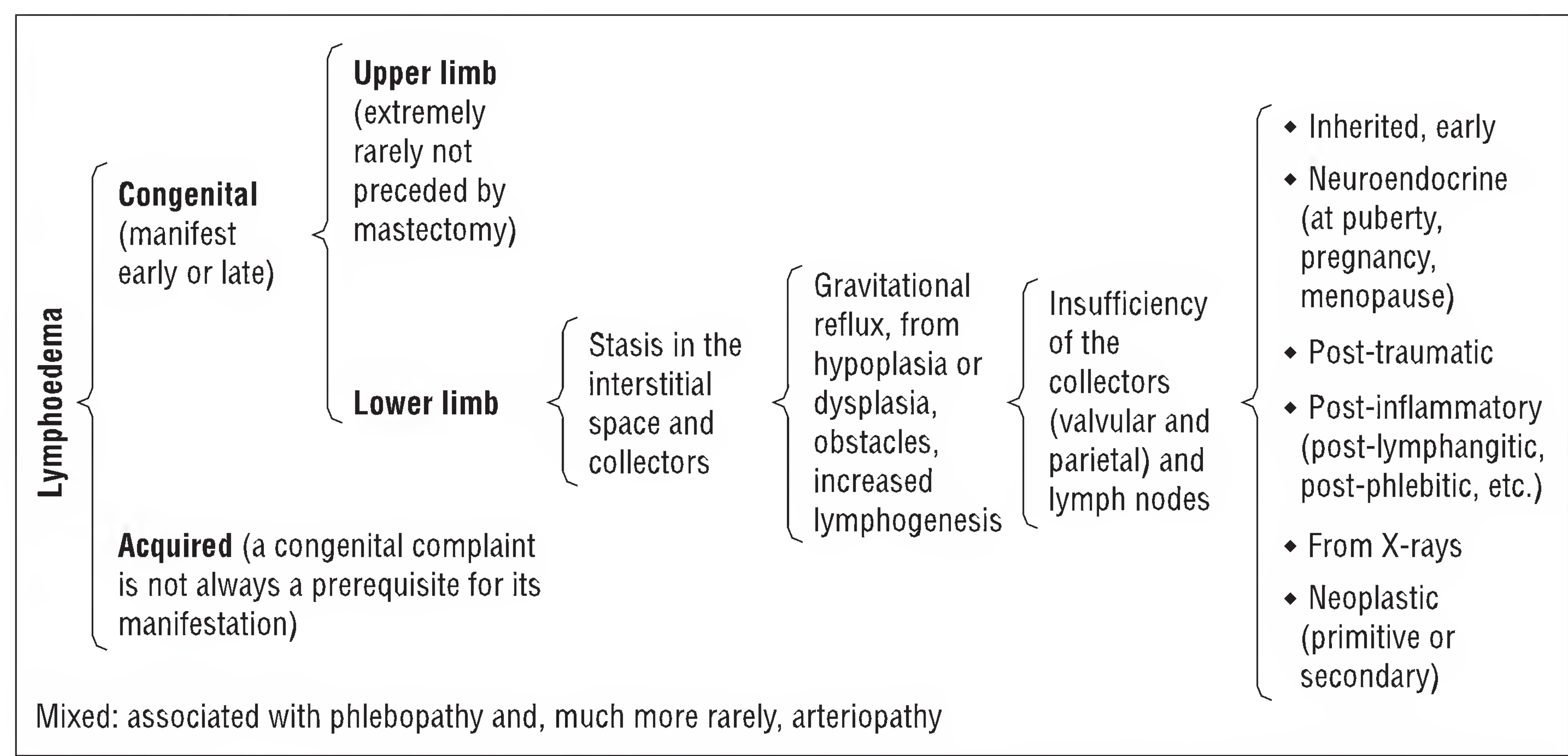


Fig. 47a.6. Classification of chronic lymphoedema of the limb according to Tosatti.

classification of lymphoedemas (Fig. 47a.6), proposed more than 30 years ago [5], still seems to be valid. **[Q1]**

Apart from some exceptional cases of acute post-lymphangitis and/or post-traumatic types, lymphoedema is normally a chronic, progressing and disabling condition characterised by a progressive volume increase of the limb(s) involved. This disease, which evolves in phases, is characterised by frequent lymphangitic, erysipeloid exacerbations with subsequent lipodermatosclerotic indurated cellulitis, chronic fibrosclerotic lymphadenitis (in primary lymphoedemas) and lymphostatic warts. The worst outcome is generally elephantiasis, with severe functional impairment or systemic sepsis. However, degeneration into lymphangiosarcoma (Stewart–Treves syndrome) is a rare sequel, most likely to occur in post-mastectomy lymphoedemas. It is important not to confuse this with cutaneous local recurrence of breast cancer. Lymphoedema of the leg can also occasionally be associated with Kaposi's sarcoma. This is not necessarily human immunodeficiency virus (HIV)-related.

The differentiation of lymphoedema from phleboedema can be based on a thorough medical history and clinical examination, paying attention to the time and conditions of onset, location, evolution, extent and volume of the oedema. Lymphoedema is hard to the touch, while venous oedema is soft and pits under finger compression. This difference reflects the underlying pathophysiology, in that stagnant lymph in the subcutaneous connective tissue is an excellent culture medium for fibroblasts. In this environment, they mature rapidly into fibrocytes, thus forming fibrosclerotic connective tissue. Lymphoedema typically begins proximally, whereas venous oedema initially affects the distal part of the lower limbs with the notable exception of phlegmasia dolens, caused by acute deep thrombophlebitis of the iliofemoral veins. Unlike phleboedema, lymphoedema does not usually evolve into dystrophic-dyschromic skin lesions or ulcers. It is more likely to be complicated by acute reticular erysipeloid lymphangitis, caused by Gram-positive cocci infection in the presence of static lymph. Phleboedema is often associated with varices and varicophlebitis, and unlike lymphoedema, it is subject to rapid postural changes and is characterised by abnormal Doppler venous flow rates with

significant increase in venous pressure when the patient is standing up. However, mixed types of lymphophleboedema also exist (as in stage III postphlebitic syndrome), with predominance of either the venous or lymphatic component. These include the complex conditions of angiodysplasia with arterial-venous hyperstomy, as seen in Mayall's syndrome [6], or congenital arteriovenous macro- and microfistulas, as seen in Klippel–Trenaunay's disease. (The latter condition is recognised by gigantism with elongation of the affected limb, varying degrees of foot dysmorphism, flat or map-like "port-wine" angioma, and hyperhydrosis of the sole of the foot.) There are also some spurious forms of lymphophleboedema, which are masked by prevailing lymphoedema and therefore more difficult to recognise. In these cases, if angiodysplasia is suspected, then routine investigations such as Doppler venous pressure measurements may be insufficient, and further investigations, including phleboscintigraphy, phlebography or digital arteriography, may be required. For the time being, lymphangioscintigraphy and conventional oil-contrast lymphography are the most suitable investigations of lymphatic and chylous oedemas. Lymphangioscintigraphy is the most popular method used in the rapid screening of lymphoedemas [7, 8] as it is a noninvasive way of imaging both superficial and deep lymphatic circulations. Since it is noninvasive, it can be repeated easily in patient follow-up, especially after microsurgery. A small tracer dose of ^{99m}technetium adsorbed in colloid spherules (colloid sulphide, rhenium, dextran) is used. The lymphotropic nature of these substances permits display of the preferential lymphatic pathways with a gamma camera, and allows measurement of the flow rate and lymph node uptake. A tracer clearance measurement is a useful parameter from a lymphodynamic viewpoint. However, lymphoscintigraphy is most useful in the study of lymphoedemas at early stages [9]. Direct lymphangiography [10] is preferred in the study of gravitational reflux lymphatic and chylous oedema of the lower limb and external genitalia before surgical intervention [11, 12]. In this examination, ultrafluid "Lipiodol" is injected into a lymphatic collector, isolated with microsurgical technique, of the dorsum of both feet. This type of investigation is minimally invasive and, if performed according to well-established standards, has minimal complications. However, rare adverse reactions have been reported. These include general complications such as pulmonary microembolism in the presence of peripheral lymphovenous fistulas or allergy to contrast medium. Local complications may also occur in the form of infection on the site of the skin incision, acute lymphangitis or lymphorrhoea. Direct lymphangiography can also be performed in children. It enables a morphofunctional study of the superficial and, with the use of proper technical support, the deep circulation [12].

Computed tomography (CT), ultrasonography and lymphangio-MRI may also provide important preoperative data on lymphatic and chylous dysfunction. Indirect lymphangiography [13] performed with dermo-hypodermic injection of a water-soluble contrast medium ("Iotasul") is useful to clarify aetiopathological aspects of primary lymphoedemas, and fluorescent microlymphography [14] can be helpful in assessing the status of the superficial dermis lymphatic web, which reflects the functional condition of the peripheral lymphatic circulation. The conventional Houdack–McMaster dye test with the injection of highly lymphotropic vital stain (Patent Blue V) is used today as a preliminary investigation in direct lymphangiography and microsurgery for a better and faster assessment of lymphatics. Recent studies by Olszewski [15] and Campisi et al. [16] have developed a system to measure endolymphatic pressure and lymphatic flow rate. These parameters, together with venous pressure assessment, help to measure the

lymph-venous pressure gradient, which is essential for a correct approach to microsurgical treatment of lymphoedemas. With this method, a lymphatic vessel is isolated and cannulated at the lower third of the leg's medial surface. Any change in the flow-pressure rate can also be recorded during microsurgery, in clino- and orthostatic positions, at rest and under dynamic conditions. These studies have shown that a valuable lymphatic-venous pressure gradient is essential to obtain medium- and long-term results by derivative microsurgery. **[Q2: B, C, E]**

Manual lymphatic drainage has been shown to be a highly effective treatment in the conservative management of lymphoedema [17–19]. This is followed by the application of bandaging and eventually graded compression stockings. The use of intermittent compression pneumatic devices is usually complementary to manual lymphatic drainage and may contribute to further reduction of the lymphoedema. Pharmacotherapy includes the use of antibiotics, particularly penicillin [20], anti-inflammatory drugs and benzopyrones [21]. The positive effect of benzopyrones was described by Casley-Smith et al. [21], but their role in the treatment of lymphoedema has yet to be clarified.

Thirty years ago, there were relatively few therapeutic solutions in the treatment of lymphoedema. Only the most severe and advanced cases of elephantiasis were treated surgically, mainly in order to reduce the volume of lymphoedematous limbs. The most popular surgical methods were those proposed by Charles [22] (total resection of skin-lipid layers), Thompson [23] (drainage with scarred subfascial skin flap), and Servelle [24] (total surface lymphangectomy). Being highly destructive and invasive operations, they could not be recommended in less advanced or initial stages or in childhood disease [25]. More recently, microsurgical lymphatic-venous and lymphnodal-venous anastomoses were introduced for the management of lymphoedema resistant to conservative treatment [26, 27]. These techniques are beneficial in not only secondary but also primary lymphoedemas [28], since early intervention is possible even in young children with some minor modifications of the technique, such as lymphatic-capsule-venous anastomosis [29].

Lymphostatic disease may be associated with venous impairment such as varices, superficial thrombophlebitis, deep venous thrombosis and postphlebitic sequelae. These conditions are a contraindication to traditional lymphatic-venous anastomosis. Therefore, novel reconstructive lymphatic surgery techniques are used [30]. These include segmental autotransplantation of lymphatic collectors [31] for the treatment of monolateral lymphoedema or the personally described method of interposition autologous venous grafting or lymphatic-venous-lymphatic plasty [32].

The use of free microvascular lymphatic or lymph nodal flaps [33, 34] is still in clinical trials. However, it opens up interesting options in the treatment of lymphoedema that fails to respond to conservative therapy and that, for congenital (aplasia or hypoplasia) or acquired (elephantiasis with diffuse obstructive lymphangitis) reasons, cannot benefit from the above-mentioned derivative or reconstructive microsurgical techniques.

Elastic stockings are worn for an average period of 1–5 years after microsurgery according to the stage of the pathology at the time of operation. These stockings aim to prevent the closure of anastomoses in the early postoperative period, following a rapid reduction of oedema and consequent decrease in lymphatic pressure and flow as a result of the microsurgical drainage [35]. **[Q3: A, B, D]**

Through a properly planned follow-up at 1, 3, 6 and 12 months, and then annually for at least the first 5 years after surgery, lymphatic microsurgery results are

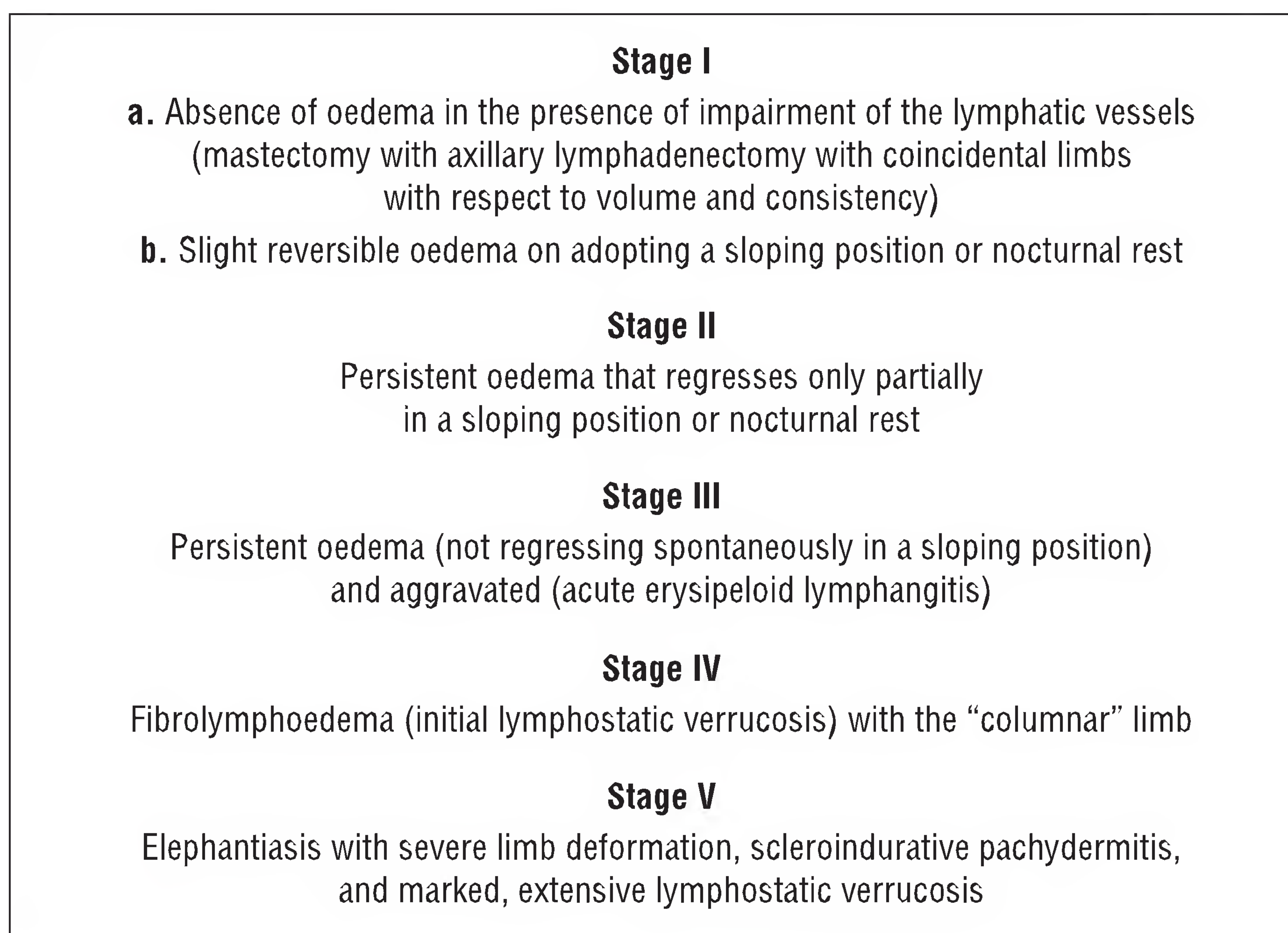


Fig. 47a.7. Clinical instrumental diagnostic staging of lymphoedema.

positive in more than 80 per cent of cases, with an even better outcome in patients operated upon precociously (at stages II and III; see Fig. 47a.7). The incidence of lymphangitic attacks decreases significantly after microsurgery. The reduction in oedema volume obtained by microsurgery is seen immediately after operation (within the first three postoperative days), and a further decrease in lymphoedema is also observed at medium- and long-term follow-up, particularly between the first and fifth years after operation. From the fifth year onwards, the clinical condition of the limb tends to remain stable with time, even more than 10 years after surgery. Lymphangioscintigraphy can document objectively that the flow through the venous graft parallels the clinical improvement over the 10-year period [36]. **[Q4]**

Turning to the prevention of secondary lymphoedema, early identification of high-risk patients (such as those undergoing oncological lymphadenectomies, particularly in combination with radiotherapy) and early diagnostic lymphangioscintigraphy [37] has been suggested [38, 39]. In these cases, early microsurgery is a reasonable option in order to fight, from their very onset, lymphoedemas that, based on a reasonable statistical probability, are expected to show unrelenting progression [40]. **[Q5]**

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47b. Management of Upper Extremity Lymphoedema with Liposuction

Håkan Brorson

A 53-year-old woman presented with an 8-year history of oedema of the right arm. Twelve years ago she underwent treatment for right breast cancer. She had mastectomy, removal of lymph glands, and postoperative irradiation. No chemotherapy was given. The arm swelling started gradually without obvious reason. Since the appearance of arm swelling she had experienced three bouts of erysipelas that were treated with penicillin. Two years ago she was treated elsewhere with combined decongestive therapy (CDT) including manual lymphatic drainage, bandaging and a set of compression garments. She had initially gained some reduction of the excess volume, but no follow-up was done.

Now her main complain was pain and problems with clothing interfering with her everyday activities. She stated that her arm was cosmetically unappealing and that she did not want to see people in public. On examination she had a swollen arm and limited limb movement.

Question 1

What would you do?

A. Perform the pitting test.

B. Perform an MRI.

C. Measure arm volumes.

D. Perform an indirect lymphoscintigraphy.

E. Start controlled compression therapy (CCT) or complete decongestive therapy (CDT) with the help of a trained lymph therapist.

The pitting test showed pitting of 2 cm (Fig. 47b.1). Arm volume measurements using the formula of the truncated cone showed an excess volume of 2700 ml. She was referred to a lymph therapist for conservative treatment (CDT) to reduce the excess volume. After 2 months the excess volume was reduced from 2700 ml to

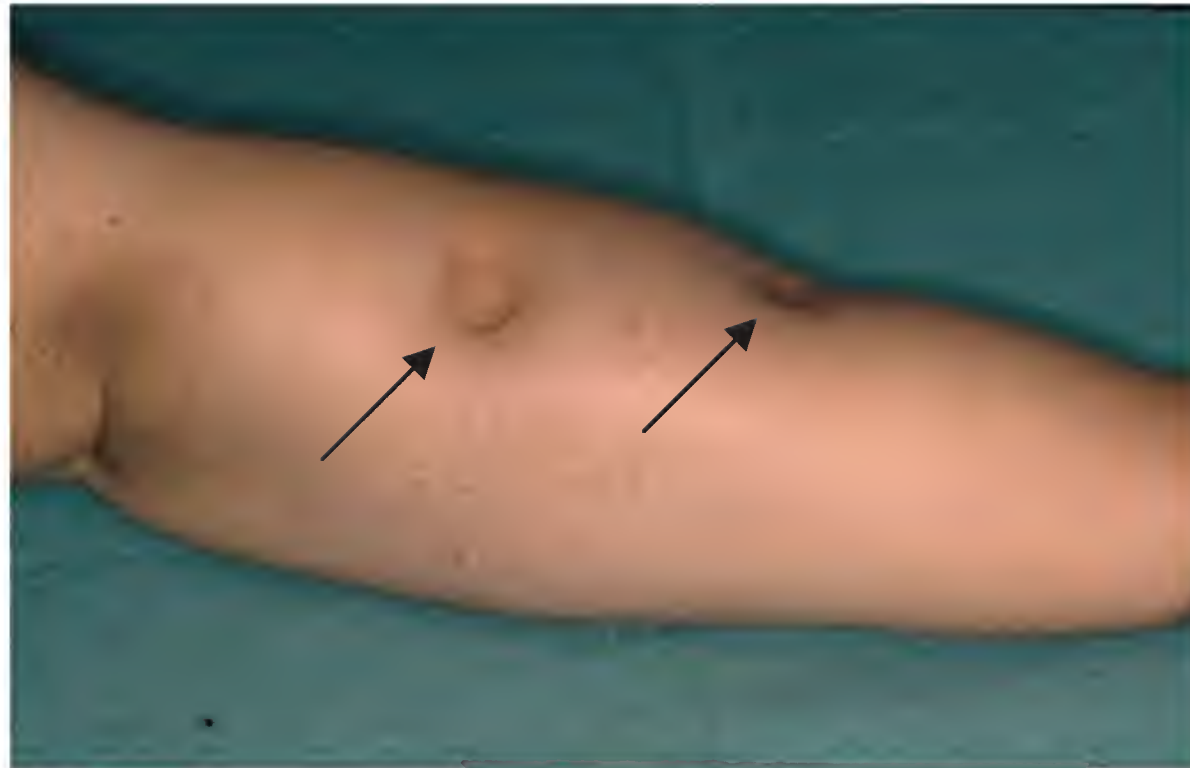


Fig. 47b.1. Marked arm lymphoedema after breast cancer treatment with deep pitting of several centimetres. The arm swelling is dominated by fluid, i.e. accumulation of lymph.

2100 ml. Further reduction was not possible in spite of a further 4 weeks' treatment. **[Q1: A, C]**

Question 2

What would be the next step in the management of this patient?

A. Perform the pitting test.

B. Perform an MRI.

C. Measure arm volumes.

D. Perform an indirect lymphoscintigraphy.

A new pitting test showed minimal pitting (4–5 mm) (Fig. 47b.2).

An MRI confirmed your suspicion of excess adipose tissue in the arm (Fig. 47b.3).

Arm volumes were calculated using the formula of the truncated cone and showed an excess volume of 2045 ml (Fig. 47b.4). The patient now wanted further reduction of the excess volume. **[Q2: A, B, D]**

Question 3

What would you do next?

A. Start conservative therapy again with the help of a trained lymph therapist.

B. Perform lymphatic-venous anastomoses.

C. Perform lymphatic-venous-lymphatic plasties (interposition autologous venous grafting).

D. Perform transplantation of lymph vessels using vessels from the thigh.

E. Refer the patient for liposuction.



Fig. 47b.2. Pronounced arm lymphoedema after breast cancer treatment. There is almost no pitting is spite of hard pressure by the index finger for one minute. A slight reddening is seen at the two spots where pressure has been exerted. The "oedema" is completely dominated by adipose tissue. The term "oedema" is at this stage improper as the swelling is dominated by hypertrophied adipose tissue and not lymph. At this stage the aspirate contains no or minimal amount of lymph (Fig. 47b.6).

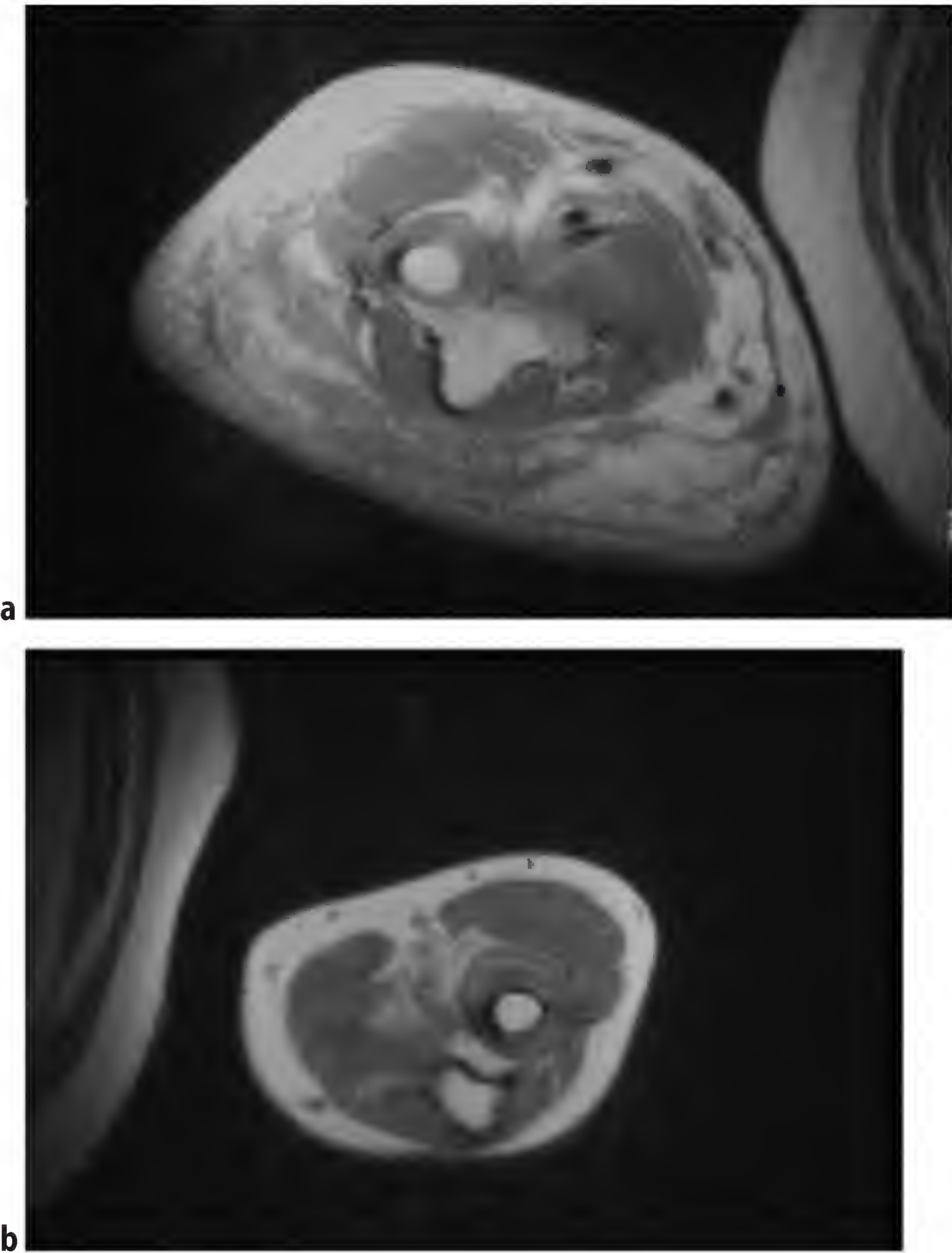


Fig. 47b.3. a MRI (elbow region) showing a right-sided, secondary arm lymphoedema after breast cancer treatment in the elbow region. Note the honeycomb pattern. **b** The healthy left side in the same patient for comparison.



Fig. 47b.4. 53-year-old woman with a preoperative oedema volume of 2050 ml in the right arm for 8 years.



Fig. 47b.5. Peroperative pictures from the beginning **a**, during **b**, **c**, and at the end **d** of surgery.

The patient was operated on with liposuction under bloodless conditions using a tourniquet (Fig. 47b.5).

After 2 hours the aspirate removed during bloodlessness measured 2100 ml. A standard compression garment was fitted on the arm before the tourniquet was released. The size of the garment was determined from preoperative measurements on the healthy arm. In order to reduce bleeding, tumescent technique was used on the most proximal part of the upped arm that was covered by the tourniquet. Tumescent technique includes infiltration with a saline-adrenaline solution (1 mg adrenaline/1000 ml saline) followed by liposuction. The next day the aspirate had sedimented and showed 92 per cent adipose tissue and 8 per cent fluid (lymph/interstitial fluid) (Fig. 47b.6).

Following surgery, controlled compression therapy (CCT) was instituted: Two days after surgery measurements were taken for custom-made compression garments, that is, sleeve and glove, two of each. The patient was discharged after 5 days, and was seen after 2 weeks. During the first 2 weeks the patient alternated two standard sets of sleeve-and-glove garments, i.e. two sleeves and two gloves. At the 2-week visit plethysmographic measurement of both arms showed an excess volume of 185 ml (91 per cent reduction). She was fitted with the custom-made compression sleeves and gloves ordered on measurements taken 2 days postoperatively.



Fig. 47b.6. The aspirate achieved under bloodless conditions usually contains 90–100 per cent adipose tissue. This picture shows a typical aspirate of 2000 ml with an adipose fraction of 90 per cent and a fluid fraction of 10 per cent.



Fig. 47b.7. Clinical result 10 years after liposuction.

During the consecutive follow-up the patient was seen at 4 weeks, 3 months, 6 months and 1 year. At the 3-month visit complete reduction was achieved and has lasted ever since. Then, as complete reduction had been achieved, she was seen once a year (see detailed information later in the text regarding CCT). At the last control, 10 years after surgery, an excess volume of –340 ml was registered; thus the treated arm was somewhat smaller than the normal one (Fig. 47b.7).

Question 4

How often, after the first year, is the patient checked when complete reduction has been achieved?

A. Every month.

B. Every 3 months.

C. Twice a year.

D. Once a year.

E. Not necessary.

Question 5

At least how many compression garments (sleeve-and-glove) a year should this patient be provided with?

A. One garment.

B. Two garments.

C. Three garments.

D. Four garments.

E. Garments are not needed.

Question 6

How long must the patient wear the compression garments?

A. Only daytime.

B. Only at night.

C. 24 hours a day.

D. 24 hours a day, lifelong.

Question 7

Who can perform the liposuction?

A. A plastic surgeon.

B. A vascular surgeon.

C. A general surgeon.

D. A dermatologist.

E. A surgeon trained especially for this kind of liposuction.

Question 8

If you have been trained to perform the surgery, but are not sure if funding for compression garments can be provided – what do you do?

A. Go ahead with the surgery.

B. Do not perform the surgery.

Question 9

In order to optimize the outcome of surgery, how do you want to arrange for the follow-up?

A. Refer the patient to a lymph therapist after surgery and tell the patient to see you in a year.

B. Set up a team with a physiotherapist and/or an occupational therapist at the clinic where you work in order to see the patient at all visits.

C. No follow-up is needed.

Question 10

Does liposuction decrease the lymph transport capacity in a patient with a chronic arm lymphoedema?

A. Yes.

B. No.

Question 11

Does liposuction with complete reduction of the lymphoedema decrease the incidence of erysipelas?

A. Yes.

B. No.

Commentary

Lymphoedema is a chronic disease with increased volume causing considerable dysfunction. Patients typically experience decreased mobility, heaviness and susceptibility to infections, and are affected by psychological problems that are secondary to the cosmetic appearance of their extremity.

Lymphoedema can be divided into various stages according to the tissue changes [1]. It can also be classified as primary and secondary. The later in life a lymphoedema appears, the more important it is to exclude other diseases, especially cancer, as the cause.

There is, so far, no cure for lymphoedema. The basis for all lymphoedema treatment is adequate compression therapy. If conservative therapy fails, liposuction can give complete reduction of the excess limb volume. To maintain this outcome, it is an absolute necessity to provide the patient with ample amounts of compression garments. It is important to measure the excess volume, as changes can be a sign of progression of the underlying disease.

Up to 38 per cent of women with breast cancer may develop lymphoedema of the arm following mastectomy, standard axillary node dissection and postoperative irradiation [2]. Limb volume reductions have been reported using various conservative therapies such as manual lymphatic drainage and compression therapy. Some patients with long-standing pronounced lymphoedema do not respond to these conservative treatments, because slow or absent lymph flow causes the formation of excess subcutaneous adipose tissue.

Liposuction removes the hypertrophied adipose tissue and is a prerequisite to achieve complete reduction. The new equilibrium is maintained through constant (24-hour) use of compression garments postoperatively. Long-term follow-up does not show any recurrence of the oedema [3].

Pitting means that a depression is formed after pressure with the fingertip on oedematous tissue, resulting in displacement of lymph into the surrounding tissue

(Fig. 47b.1). In order to standardise the pitting-test, one presses as hard as possible with the index finger, for 1 minute, on the region to be investigated. The amount of depression is estimated in millimetres.

Arm volumes can easily be measured by circumferential measurements every 4 cm along the arm according to Kuhnke [4]. In our clinic we use plethysmography (i.e. water displacement technique), which is considered the golden standard . Both arms are always measured at each visit, and the difference in arm volumes is designated as the oedema volume [5, 6]. The decrease in the oedema volume is calculated as a percentage, thus:

$$\frac{(OA_{pre} - HA_{pre}) - (OA_{post} - HA_{post})}{OA_{pre} - HA_{pre}} \times 100,$$

where
OA_{pre} = oedematous arm before treatment
HA_{pre} = healthy arm brfore treatment
OA_{post} = oedematous arm after treatment
HA_{post} = healthy arm after treatment

Arm volume measurements for calculating the oedema volume are taken at each visit on both arms.

Oedema dominated by hypertrophied adipose tissue and/or fibrosis shows little or no pitting (Fig. 47b.2). *Stemmer's sign* implies that one with difficulty, or not at all, can pinch the skin at the base of the toes or fingers. This is due to increased fibrosis and is characteristic of lymphoedema.

Indirect lymphoscintigraphy is not necessary in secondary lymphoedema, but is recommended in patients with an excess volume where the diagnosis is unclear.

In healthy subjects the rate of blood flow and lymph flow through adipose tissue is inversely related to its growth, and a slow flow rate is considered to be a factor promoting lipogenesis and further deposition of fat. This process is enhanced by the transformation of macrophages into adipocytes [7]. This may explain the marked hypertrophy of the adipose tissue seen in patients with chronic lymphoedema (Fig. 47b.6) [8]. In late stages subcutaneous lymphoedema becomes firm and denser and is dominated by adipose tissue hypertrophy, and pitting is usually less pronounced or sometimes absent (Fig. 47b.2). An additional factor that may play a role in increasing the amount of adipose tissue is chronic inflammation present in long-standing lymphoedema. This phenomenon is also seen in patients with Crohn's disease where the inflamed intestine is surrounded with excess adipose tissue ("fat wrapping") [9]. Probably pinocytosis of white blood cells, in combination with activation of fibrocytes, increases the connective tissue component of the primordial loose subcutaneous fat [10].

Despite prophylaxis, the lymphoedema will often progress slowly but steadily, producing a variety of symptoms as described in our patient. Surgical treatment is indicated in patients who fail to respond to conservative treatment [7, 8, 10].

Various surgical procedures have therefore been proposed to reduce lymphoedema, including debulking procedures with skin grafting and omental transposition [11–19]. None of these methods gave satisfactory or long-lasting results.

The breakthrough in reconstructive microsurgery has stimulated renewed interest in the management of lymphoedema. During the last decades, anastomoses have

been established between lymph nodes [20] or lymph collectors [21, 22] and the venous system. Promising results have recently been reported after transplantation of lymph collectors [23, 24], as well as after the creation of various forms of lymphatic venous anastomoses [25, 26].

Even if the microsurgical methods are attractive from a physiological point of view, they do not give consistently satisfactory results. Patients need to wear compression garments after surgery, indicating that normal lymph transport has not been achieved. Complete reduction cannot be achieved in patients with a long-standing non-pitting lymphoedema because the hypertrophied adipose tissue remains unaffected by the microsurgery. A surgical approach, with the intention to remove the hypertrophied adipose tissue, seems logical when conservative treatment has not yielded satisfactory oedema reduction and the patient has not experienced symptomatic relief. **[Q3: E]** This condition is especially seen in chronic, large arm lymphoedemas around one litre in volume, or when the volume ratio (oedematous arm/healthy arm) is approximately 1.3. There is no upper age limit in order to be considered for liposuction, but active tumour disease and ulcerations are contraindications. The detailed surgical technique has been reported [3, 6, 27, 28].

Postoperatively the controlled compression therapy (CCT) is crucial, and its application is therefore thoroughly described and discussed at the first clinical evaluation. If the patient has any doubts about continued CCT, she is not accepted for treatment. After institution of the compression therapy, the custom-made garments (Jobst® Elvarex BSN medical, compression class 2, rarely class 3) are taken in at each visit, using a sewing machine, to compensate for reduced elasticity and reduced arm volume. This is most important during the first 3 months when the most notable changes in volume occur. At the 1-month visit two new custom-made compression garments are measured for. This is repeated at 3, 6, (9), and 12 months. It is important, however, to take in the garment repeatedly to compensate for wear and tear. This requires additional visits in some instances, although the patient can often make such adjustments herself. When the oedema volume has decreased as much as possible and a steady state is achieved, new garments can be prescribed, using the latest measurements. In this way, the garments are renewed at least four times during the first year. Two sets of sleeve-and-glove garments are always at the patient's disposal; one being worn while the other is washed. Thus, a garment is worn permanently, and treatment is interrupted only briefly when showering and, possibly, for formal social occasions. **[Q6: D]** The patient is informed about the importance of hygienic measures and skin care [3, 6, 27, 28].

The life span of two garments worn alternately, is usually 4 to 6 months. After complete reduction has been achieved, the patient is seen once a year, when new garments are prescribed for the coming year, usually four garments and four gloves (or four gauntlets). **[Q5: D]** Patients without hand oedema can usually discontinue the glove or gauntlet after the first year. In very active patients three to four pairs of sleeve-and-glove garments a year may be needed. In very young, active patients sometimes two sleeve-and-glove garments every month must be prescribed. It is like when prescribing insulin. During the first postoperative year the correct amount is estimated. If too few, the volume will increase. One must remember that a compression garment is not a static tool and should be considered as perishable goods.

Liposuction is preferably performed by plastic surgeons. The technique used for lymphoedema is quite different from that used in cosmetic surgery, so even a plastic surgeon must learn this approach for lymphoedema. Any interested surgeons,

under careful observation, can therefore learn the technique in order to treat lymphoedema. **[Q7: E]**

If funding cannot be achieved for ordering the proper amount of garments, then surgery – or for that matter any kind of lymphoedema treatment – should not be performed. **[Q8: B]** It is a waste of time and money. It is like sitting in a leaking boat, scooping out water all the time instead of putting a plug in the leak. The compression garment is the safe plug and the weakest link in lymphoedema treatment. So, a prerequisite to maintain the effect of liposuction is the continuous use of a compression garment.

The already decreased lymph transport capacity is not further impaired by liposuction [29]. **[Q10: B]** Liposuction decreases the incidence of erysipelas. **[Q11: A]** The point of bacterial entry may be a minor injury to the oedematous skin, and impaired skin blood flow may respond inadequately to counteract impending infection. Reducing the oedema volume by liposuction increases skin blood flow, and probably decreases the reservoir of proteinaceous fluid and adipose tissue, which may enhance bacterial overgrowth [30]. Through the combination of liposuction and CCT the lymphoedema can be completely removed. Long-term follow-up (7 years) does not show any recurrence of the oedema [3, 27, 31].

In our unit a team that consists of a plastic surgeon, an occupational therapist and a physiotherapist assesses patients with lymphoedema. **[Q9: B]** A 60-minute period is reserved for each scheduled visit to the team, when arm volumes are measured, garments are adjusted or renewed, the social circumstances are assessed, and other matters of concern are discussed. The patient is also encouraged to contact the team whenever any unexpected problems arise, so that these can be tackled without delay. A team approach such as the one described seems to be ideal in providing the patient with a fully informed consent for an intervention, and for successful maintenance of immediate postoperative results. The team also monitors the long-term outcome, and our experiences so far indicate that a visit once a year is necessary to maintain a good functional and cosmetic result in most cases after complete reduction. **[Q4: D]**

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